Journal
OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

AVMA Convention—San Antonio, Oct. 15-18, 1956

Contents continued on adv. pages 2 and 4

Vol. 129 **October 1, 1956** **No. 7**

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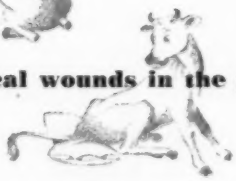

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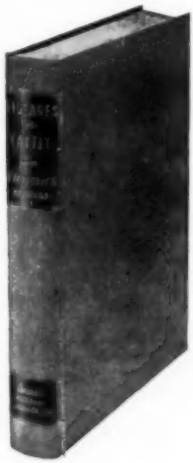

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AVMA ☆ Report

Veterinary Medical Activities

✦ President Floyd Cross attended the Oklahoma A. & M. Conference for Veterinarians at Stillwater on September 27-28.

* * *

✦ President-Elect Wayne O. Kester participated in the meeting of the New Mexico V.M.A. held at Portales on September 11-12; he also attended the opening of the Plum Island Laboratory during the week of September 23.

* * *

✦ Executive Secretary J. G. Hardenbergh and Dr. F. A. Todd, assistant administrator, ARS, U.S.D.A., attended the meeting of the National Advisory Council on Rural Civil Defense held at Battle Creek, Mich., on September 13-14.

* * *

✦ Washington Representative J. A. McCallam and Dr. J. W. Cunkelman, member of the AVMA Committee on Food Hygiene, attended the meeting called by the Food and Drug Administration on "Public Health Significance of Antibiotics in the Milk Supply" held in Washington, D.C., September 10.

* * *

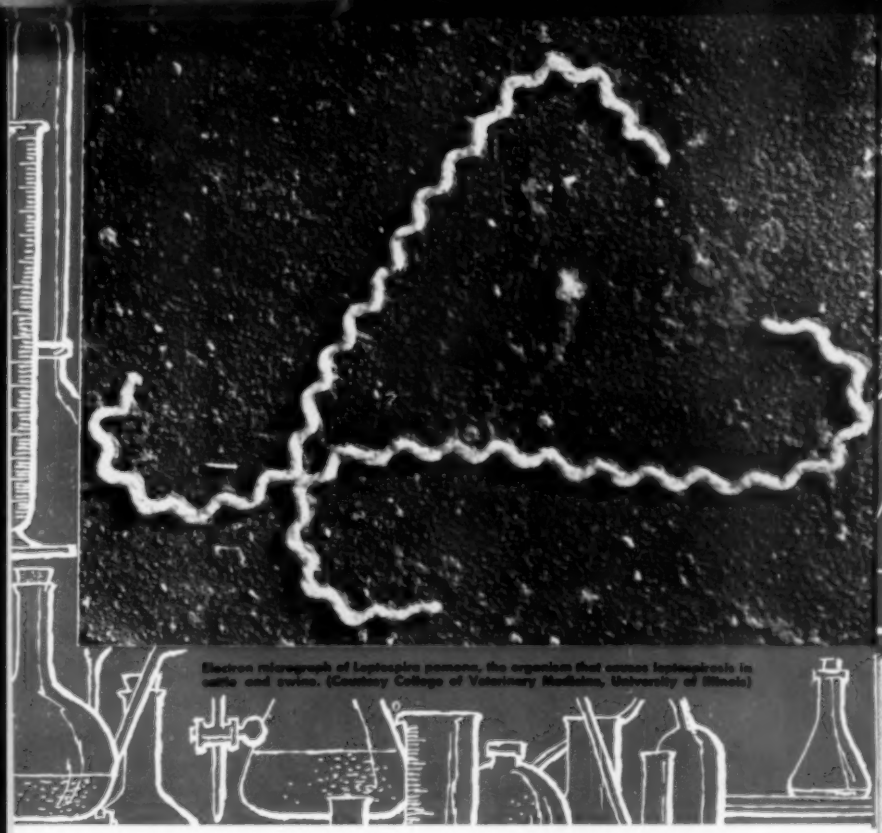
✦ Dr. C. H. Pals, Washington, D.C., and Colonel W. E. Jennings, U.S. Army, Europe, participated in the first Symposium of the International Association of Veterinary Food Hygienists, held in Utrecht, Netherlands, August 27—September 1. Other members from the United States participating in this program included Colonel Rowland W. Rushmore, Dr. G. S. McKee, and Dr. R. D. Barner.

* * *

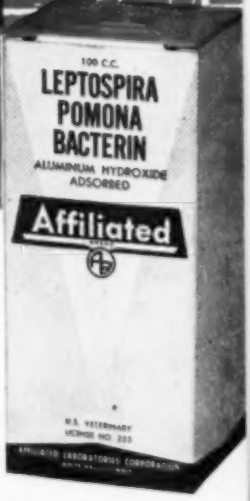
✦ Lt. Colonel N. A. Lasher, staff veterinarian, U.S.A.F., Europe, and Dr. R. C. Klussendorf, Terre Haute, Ind., represented the Association at the Seventy-Fourth Annual Congress of the British Veterinary Association held at Leamington Spa, England, September 16-22.

* * *

✦ About 42 officers of the Army and Air Force Veterinary Corps, who attended the Meat and Dairy Hygiene School at the Quartermaster Depot, Chicago, visited AVMA headquarters on September 28. Members of the AVMA staff spoke to the group on the organization and activities of the Association and other professional matters.



Electron micrograph of Leptospira pomona, the organism that causes leptospirosis in cattle and swine. (Courtesy College of Veterinary Medicine, University of Illinois)



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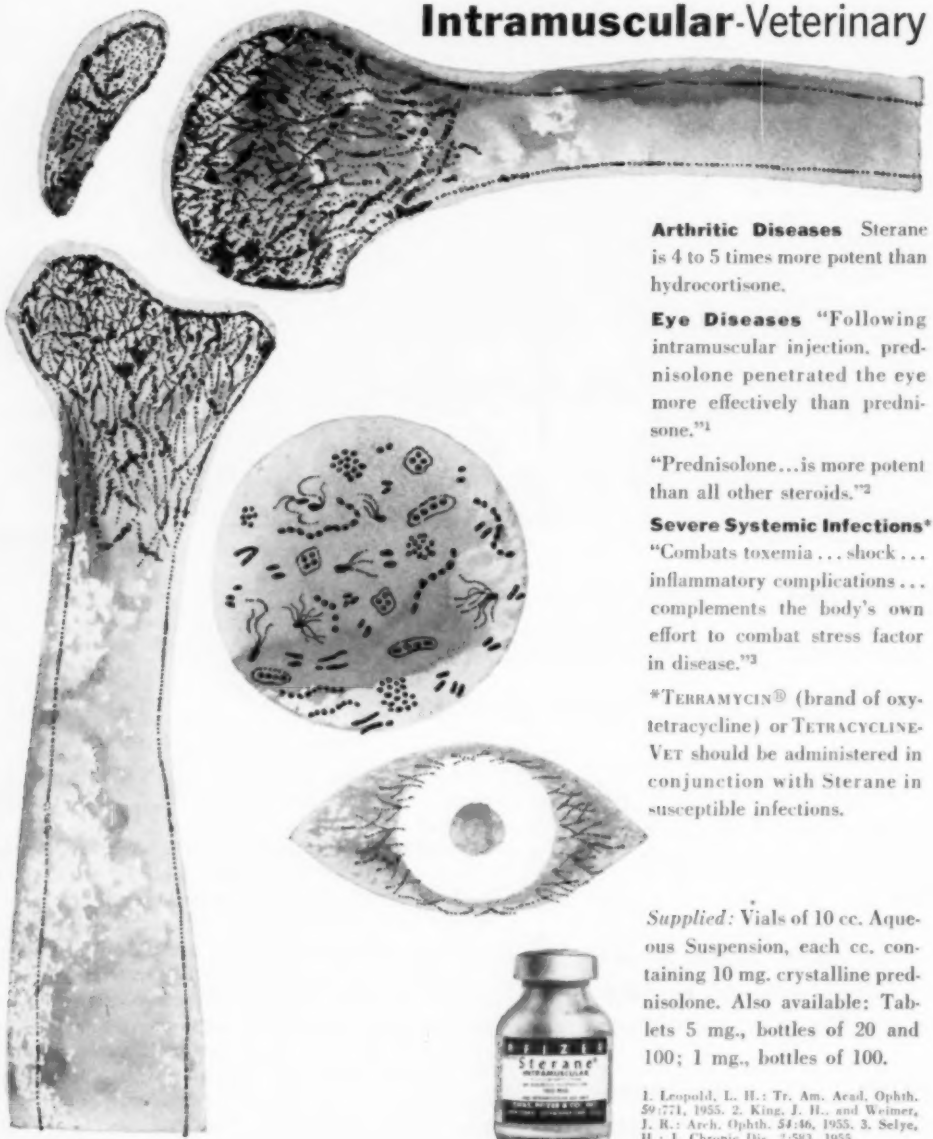

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1. Leopold, L. H.: Tr. Am. Acad. Ophth. *59*:771, 1955. 2. King, J. H., and Weimer, J. R.: Arch. Ophth. *54*:46, 1955. 3. Selye, H.: J. Chronic Dis. *2*:583, 1955.

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Vol. 129 October 1, 1956 No. 7

Oral Rehabilitation of Dogs

C. E. BILD, D.V.M.
Miami, Florida

THIS PAPER deals with nutritive needs in oral rehabilitation of dogs.

Schoenheimer[1] observed that all body cells are in a state of flux. Generally, every cell undergoes anabolic and catabolic changes every day. Added catabolism accompanies elevated temperature. Primary and secondary infections cause added catabolism, but the number, extent, combinations, and causes of catabolic factors are limitless.

Oral rehabilitation or oral alimentation is useful whenever an animal is partially or completely off feed or is losing weight or hemoglobin level, or both. Usually, one can not, with oral alimentation alone, completely offset the catabolism of medical or surgical problems. Nevertheless, my clinical experience has shown that added nutrients are worth more than the time and effort. They may well be the deciding factor in the final outcome of a specific problem.

INFECTIONS

Acute nutritive deficits occur in acute infections of dogs. When a virus or any type of infection invades the cells, the result is added catabolism. Hypothetically, if an animal consumes its regular ration but has a virus infection or an elevated temperature, or both, a nutritive deficit occurs. If the animal is partially off feed, the catabolism continues and the nutritive deficit increases. This is particularly important in the dog with distemper that is completely off feed and shows an elevated temperature. However, these factors are of little clinical importance unless the animal has been off feed more than two days.

These precepts are true of the mature animal. The growing animal has greater needs than the mature one; therefore, the over-all deficit in the young, ill animal is greater.

NEPHRITIS

Chronic nephritis is another cause of chronic nutritive deficit in the mature animal. This condition causes wastage of body essentials which occur, perhaps, over a long time. As Homer Smith said, "We are not what we eat, but what the kidneys keep." The animal that loses (protein) albumin in the urine, often has a low serum albumin. However, the total serum protein may be high. One may find abnormally high serum globulins as compensatory factors, thus reversing the normal ratios of albumin to globulin. Some degree of anemia or weight loss, or both, may be observed, and the animal may have a poor coat. In the dynamics of equilibrium, the animal with chronic nephritis may adjust itself to a low hemoglobin level, but it is a mistake to think of a low hemoglobin level as being normal. This is similar to thinking that only small matches are safe around gasoline.

When body essentials have been wasted, permanent relief must be sought with oral nutrients. Often this is a slow process and some animals show no improvement.

Just as an old automobile may run as satisfactorily as, but less efficiently than, a new one because it needs more oil, gasoline, and frequent check-ups, the animal with nephritis is less efficient in its dietary essentials than is the well animal. Since the albumin lost in the urine is a complete protein, the need is for complete proteins such as meat, milk, and cooked eggs.[2] Dry skimmed milk is an excellent protein supplement.

When the nutrients allow for maintenance, deficits, and wastage, many of these animals live a normal life span and look well to the casual observer. The greatest

Dr. Bild is a small animal practitioner in Miami, Fla.

misfortune of the well-looking nephritic animal is that it has little margin for stress situations.

PARASITES

A third cause of a chronic nutritive deficit may be parasites. Apparently, no toxins are formed by hookworms, but they cause a loss of blood and iron. The actual deficit caused by the parasites is proportional to the nutritive intake. By adding iron and additional nutrients, the clinician may maintain both the host and the parasites in an excellent state of nutrition, for the better the nutrition of the animal, the less the problem of hookworms and, also, coccidia. Subclinical dietary intakes and imbalances cause and complicate countless disease problems.[3] A nutritive deficit occurs in any animal that does not receive all the essential amino acids at each feeding.[4]

SURGERY

Another cause of acute nutritive deficits is surgery. Essential amino acids are lost in proportion to the extent and stress of the surgery.[5] Other body essentials are also lost because of stress.[6] However, no clinical significance is attached to the nutritive deficits involved in simple operations such as oophorectomy and removal of small tumors with local infiltrations.

In mature animals, nutritive deficiency is often involved in the over-all problem of pyometra or perineal hernia. After anesthesia and surgery, the immediate resuscitative problem is solved by the use of antibiotics, water-electrolytes-dextrose, and often whole blood infusion. At this point, however, many of these animals are in low-grade medical shock caused chiefly by nutritive deficits and absorption of toxins. Part of the deficit may be attributed to the nutrients lost as endometrial exudate. The fact that the animal is almost always off feed increases the deficit. To make matters worse, many such animals have the added disability of chronic nephritis, with resultant loss of protein in the urine. Tissue fragility is common. After surgery and immediate resuscitation, the principal problem is nutritive intake. This is important in recovery.

FRACTURES

Over-all stress and trauma factors are involved in fractures. The reason for the swelling at a fracture site is the accumulation of plasma or whole blood. When red blood cells leave the circulatory tree in fractures, or when blood is lost into the peritoneal or pleural cavities, a nutritive deficit results, its seriousness depending on the extent of the hemorrhage. When these red blood cells are absorbed, they are deaminated and excreted as nitrogen in the urine.[7] For this reason, intraperitoneal, subcutaneous, or intramuscular blood infusion is not proper.

In general, it may be said that catabolism results from *any* stress situation. The nutritive loss is in ratio to the extent and duration of the stress and the resistance of the animal.

REPLACEMENT THERAPY

An analogy can be drawn between nutrients in the body and a bank account. If one withdraws a large amount of money from his checking account, he probably deposits funds later to replace the withdrawal. Continued withdrawals without counterbalancing deposits will, in time, cause some painful difficulties. Toxic destruction of protein[8] is probably excessive catabolism (withdrawals) and insufficient anabolism (deposits). A parasitized dog, with a severe fracture and trauma superimposed over a distemper infection, may show strikingly extensive catabolism and there may be only minor constructive metabolism.

In clinical practice, we see many cases wherein the problem of nutritive needs is the chief part of treatment of an ill animal. Here is the procedure we follow: For a 20- to 25-lb. dog, we frequently prescribe the administration, eight to 12 times daily, of 1 teaspoon of table sugar, 1 teaspoon of dry skimmed milk, 1 teaspoon of water, and ½ teaspoon of myeladol.®* This provides 10 or 15 calories per pound of animal weight in addition to any other nutrients the animal will take.

If there is a long-standing nutritional deficit, in which this alimentation might continue for a week or more, or if there is extensive acute catabolism, the animal may be given the sugar, milk, and myeladol, as described, every hour around the clock. To each of these forced feedings may be added a pinch of yeast powder, a pinch of wheat-germ meal, and ½ teaspoon of salad oil. Yeast contains a high amount of potassium[9] and this helps to replace the potassium loss which is, at least, in proportion

*Myeladol is produced by the Upjohn Co., Kalamazoo, Mich.

to weight and protein loss.[10] The forced feedings may be increased to about 40 calories per pound daily.

Actually, in the usual clinical problem, this diet is not sufficient to completely balance the catabolism of illness, although it may well be the deciding factor in the final outcome.

When this special feeding is continued for a week or more, the urine is checked frequently. If we find the urine chlorides low in relation to the specific gravity, we add an oral electrolyte product, lytren,† to the feeding. A 1-oz. jar of lytren is given daily to a 20- to 25-lb. dog in the divided oral feedings. We have increased this dosage on occasion without causing vomition. A specific case was a Dachshund with pelvic fractures, which also had an extremely large cystic calculus and extensive contusion to the abdominal wall that was visible externally. The calculus was removed a week after the fracture occurred, and the abdominal contusion was visible internally. We attempted to give the dog as much food as possible, feeding four times daily, and also gave the multiple oral feedings. In this case, we used two thirds of a 2-oz. jar of lytren daily. Recovery was uninterrupted and satisfactory.

Evaluation of a low urine chloride level has many pitfalls. Low levels of urine chlorides may result from depletion of salt. On the other hand, they may occur because the canine kidney, due to stress or renal dysfunction, or both, has a lowered chloride output in relation to specific gravity. There is also the problem of minimum requirements and maximum tolerance.

Vomiting is rare in these oral routines. A good percentage of animals will take the feedings voluntarily. If loose stools occur, the clinician may find it necessary to adjust the dosages.

PUPPIES

Modified procedures are followed for immature animals. The thin, dehydrated puppy, 8 weeks old, with poor appetite and diarrhea is a common clinical problem. This puppy probably needs every known nutrient. If heavily parasitized, he is treated immediately. Often a small puppy will need at least 12 cc. per pound of electrolytes and water, given subcutaneously. (Sugars should not be administered subcutaneously or intraperitoneally.)[11,12] Distemper serum and antibiotics are indicated.

Despite the salutary effects of these routines, the puppy is still not properly hydrated and is far from being well. The object is to get him to eat satisfactorily four times a day. Because we do not like to hospitalize puppies, we ask the owner to give them, several times daily (assuming he weighs 6 lb.), ¼ teaspoon each of table sugar and dry skimmed milk, a few drops of water, and approximately 8 drops of myeladol. Frequently we find that the puppy will start eating satisfactorily within a day or two. The feeding routine may then be changed to six times daily for a few days. There is more to the dehydration problem than a deficit of water and electrolytes. Often the puppy will not be severely dehydrated on the second day of treatment.

Our clinical experience indicates that low hemoglobin levels and general unthriftiness in puppies is more closely related to poor feeding than to parasites. In these instances, we find principally a lack of quality and quantity of proteins in the ration.

TRAUMA

Severe trauma is another phenomenon that calls for oral rehabilitation. We start the feeding routines as early as possible and continue them until the animal is well on full feed. The need for oral rehabilitation is controlled by the extent of the injury. In trauma—as in acute infections, distemper, chronic nephritis, leptospirosis, parasites, involved surgery, and diarrhea in puppies—the clinical need for oral rehabilitation depends on the severity of the stress that is causing metabolic imbalance. In addition, the clinician must consider the extent of the nutritive deficit, which may be of short duration (as in puppies with diarrhea for 2 days), or which may be of long duration (as in chronic nephritis), or which may be both acute and severe (as in some fracture trauma situations or in severe leptospirosis).

VOMITION

In cases of simple regurgitation, when the cause is poor management, the recommended procedure would call for the feeding of raw lean beef only, four times daily for a few days. This may be followed by equal portions of raw ground beef and canned dog food three or four times daily for a few days. The adult, chronically

†Lytren is produced by Mead-Johnson.

vomiting Boston Terrier or Boxer often tolerates neither fats nor sweet or canned milk in routine feeding. However, most of them will tolerate the hourly administration of table sugar, dry skimmed milk, and myeladol, if the need should arise.

RAW BEEF DIET

The puppy that has had diarrhea for two or three days may be fed raw beef four to six times daily for a day or two. The same routine is recommended for the dog that eats grass, then regurgitates and passes blood in the stool.

Careful clinical observation shows there is no contradiction in feeding good lean beef as soon as possible after any type of surgery, including operations on the intestinal tract. In surgical repair, the sutures will be strongest on the first day of the operation. The day following removal of the anal glands, we send the animal home and always prescribe raw beef, only, for a week or ten days, followed by equal portions of raw beef and the regular ration for a few days.

Before elective surgical procedures on puppies 3 to 4 months old, lean ground beef is prescribed at midnight and at 5 a.m. before the animal is presented at the hospital for anesthesia at 9 a.m.

RE-EVALUATION OF ROUTINES

The clinician should continually re-evaluate oral rehabilitation routines with the thought of finding ways to improve them. This can be done by daily observation of the progress of the individual by: (1) a clinical appraisal of the general well-being of the animal; (2) checking weight; (3) checking hematocrit or hemoglobin; and (4) urinalysis if the animal is mature.

As a test of the routines described in this article, we have maintained 3 Pointer puppies, 4 months old, with these oral rehabilitation routines as the only source of nutrients for two weeks. The puppies kept both their weight and hemoglobin level and were active.

Oral rehabilitation may be used any time an animal is off feed, or is partially on feed, but is losing weight or hemoglobin level, or both. Multiple oral administration of table sugar, dry skimmed milk, etc., is a valuable clinical aid. Nature has provided a special adaptability in oral alimentation; thus, selective absorption rectifies many of the imbalances administered.

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Committee on Laboratory Animals

On Sunday, Oct. 14, 1956, the Committee on Veterinary Care of Laboratory Animals will meet at 1:30 p.m. in the Oriental Room, Gunter Hotel, San Antonio, Texas.

The program will include the following speakers and their subjects: Carl F. Schlotthauer, Mayo Foundation, Rochester, Minn. (Care and Housing of Dogs Used in Research); Maurice W. Hale, Walter Reed Army Medical Center, Washington, D.C. (Disease-Free Animal Colonies); William I. Gay, National Institutes of Health, Bethesda, Md. (Quarantining and Conditioning Monkeys for Use in N.I.H.); and Lisbeth M. Kraft, Berg Institute Scientific Services (Diarrhea of Infant Mice).

Case Reports at Meeting of Zoo Veterinarians

The eleventh annual meeting of the zoo veterinarians group will be held during the AVMA convention in San Antonio, Texas, on Tuesday, October 16, at 2:00 p.m. in the Alamo Room of the Gunter Hotel.

Members who wish to present case reports on zoo animals at this meeting should contact Dr. Patricia O'Connor, Staten Island Zoo, Staten Island 10, N.Y.

Kodiac bears have been successfully kept away from salmon-breeding streams by the use of electrical fences.—*Sci. News Letter, Aug. 11, 1956.*

The Treatment of Tetanus with Curare—A Little Known Chapter in Veterinary History

J. F. SMITHCORS, D.V.M., Ph.D.

East Lansing, Michigan

THE RECENT report of Booth and Pierson[1] on the use of a pure curare alkaloid in the treatment of tetanus in a horse brings to mind an obscure incident in the history of veterinary medicine. While their mention of the first use of curare in the treatment of tetanus in man is substantially correct, it gives little indication of the great interest in the subject which lasted several decades. According to McIntyre,[2] curare was used (unsuccessfully) in 1858, after which there were more than 30 articles on the use of curare in tetanus recorded to the early 1880's. McIntyre states that these reports "may be summarized very readily; they do not, either singly or collectively, present proof of the drug's value in this disease."

Physicians apparently became disenchanted with curare in tetanus, for the drug was not used again, to any extent, until almost 50 years later. As a result of the finding in 1944 that prolonged complete curarization in dogs resulted in death even though respiration was artificially maintained, McIntyre, whose book on curare deserves careful reading, recommends that curare be used only to control spasm, as Booth and Pierson did in their work. On the other hand, "heroic doses and artificial respiration should be used in those desperate cases that are otherwise almost certain to die."

FIRST USE OF CURARE

McIntyre states,

> The first to use curare in tetanus appears to have been Sewell . . . who (in 1835) treated two cases of equine tetanus with curare. Sewell wrote: "To effect restoration from suspended animation by woorara (curare) requires about four hours of artificial respiration, to be kept up with great regularity. Neither of the animals on which I tried it died from the experiment, or the return of tetanus: but one from inanition, and the other from repletion."

This provides, in modern times, a rare instance recognizing the fact that the treatment of tetanus with curare was first tried on the horse. From the viewpoint of veterinary history, however, McIntyre's brief mention fails to bring to light a most interesting, if a bit disappointing, facet of the subject.

The British veterinary historian, General Sir Frederick Smith,[3] relates the tragic overtones of Sewell's relation to the veterinary profession during its infancy in England. William Sewell was apprenticed at the age of 15 to Edward Coleman, the autocratic surgeon-professor of the London Veterinary College, and received his diploma in 1799. He was then made Coleman's assistant and, later, assistant professor. His entire life was spent, literally, within the confines of the college walls. Neither Coleman nor Sewell were popular with members of the veterinary profession, including many of their own graduates but, until Coleman's death in 1839, both were on good terms with a group of medical men who dictated college policies. Apparently, as a consequence of Sewell's aloofness from the veterinary profession, he communicated his original work, including the discovery of the central canal of the spinal cord of the horse, to his medical friends. This not only deprived the veterinary profession of the reports of his worthy investigations but, because of the generally deprecatory attitude taken by many medical men toward veterinarians at the time, his work found only moderate recognition in medical circles. Thus, not only is Sewell virtually unknown in veterinary history but, because his work is reported primarily in the medical literature, the veterinary significance of his work is also largely unknown, or at least not recognized. His failure to provide veterinarians with the details of his work on the use of curare may, in part, account for the failure of veterinarians to capitalize on this excellent original research.

The eminent veterinarian, William Youatt, editor of the *Veterinarian* (London), was properly incensed over Sewell's snub of his professional colleagues. Writing in his editorial capacity in 1835, Youatt,[4] "in no unfriendly voice," asked:

> What does the worthy Professor mean by this strange mode of publication? Is he not aware that,

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in virtue of the office which he has the honour to fill, every discovery which he makes, every improvement on our art which may suggest itself to his mind, is the property of the profession? . . . .

Some annoying circumstances must occasionally attend this mode of publication. The following story will afford an illustration of this: A professor of high standing in one of the metropolitan medical schools a few days ago asked the writer (Youatt) . . . to assist him in performing (on a rabid dog) Mr. Sewell's experiment on a tetanic horse, *viz.* to destroy it by the woorara poison, to resuscitate it by artificial respiration, and to see whether, as in the horse, the nervous erythrism had been completely got rid of. The veterinarian (Youatt) stared at him with astonishment, and said he was not aware of such an operation having been performed . . . and told him . . . he must have been misinformed. "Oh, no," was the reply: "another surgeon and I had it from Mr. Sewell himself, and he gave it to us in print."

Thus was an operation, the conception of which did Mr. Sewell credit, and the success of which would have immortalized him—a truly glorious operation—in a manner lost to the profession.

That Sewell's experiment excited some interest among medical men may be adduced from an account of his work which appears in Watson's *Physic* (1845), an extensive series of lectures delivered by Thomas Watson, M.D. at King's College, London. Watson, a close friend of Youatt, speaks highly of the veterinary profession. In writing of tetanus,[5] he mentions that a Mr. Morgan had produced artificial tetanus by inserting a poison from Java into a wound, relieving the tetanic symptoms with a North American poison.

Professor Sewell, of the Veterinary College, has tried this principle on one case at least, where tetanus was the result, not of any poison, but of disease. Not having had an opportunity of getting the particulars of this case from Mr. Sewell himself, I give you Mr. Mayo's (a surgeon) account of it.

'A horse, suffering from a severe attack of tetanus and locked-jaw, the mouth being too firmly closed to admit the introduction of either food or medicine, was inoculated on the fleshy part of the shoulder with an arrow point coated with wourali poison (curare). In ten minutes apparent death was produced. Artificial respiration was immediately commenced, and kept up about four hours, when reanimation took place. The animal rose up, apparently perfectly recovered, and eagerly partook of corn and hay. He was unluckily too abundantly supplied with food during the night. The consequence was over-distension of the stomach, of which the animal died the following day, without, however, having the slightest recurrence of tetanic symptoms.'

I had fancied that the death had resulted from some injurious effect upon the lungs, produced by the artificial breathing. But I have little doubt that Mr. Mayo derived his statement from Mr. Sewell himself. The experiment deserves to be carefully repeated.

Both of Sewell's cases are reported in the *Veterinarian*[6] for 1858, his second case being that of an ass "labouring under an attack of the severest form of tetanus." The report continues,

The animal was in a very emaciated state . . . and being unable to walk, he was conveyed in a barrow. The Wourali was employed as in the former case, with the same effect, and artificial respiration produced reanimation in about the same time. . . . [There was not] a sufficient recovery of strength to enable the animal to rise; nevertheless the disease had entirely disappeared, and for twenty-seven hours he was enabled to take a little food; at the end of that time he died without having shown a single symptom of tetanus subsequent to the inoculation of the poison.

MEDICAL APPLICATION

Medical application of Sewell's work apparently remained largely experimental. Waring's *Therapeutics*[7] (London, 1871) states merely, "It has been proposed as a remedy in hydrophobia, tetanus, and some other nervous affections, and as an antidote to strychnia." McIntyre states that curare was first used for chorea in 1878, for epilepsy in 1860, and for strychnine poisoning in 1905, but that there is no evidence that curare was ever actually used for hydrophobia in spite of suggestions in 1878 that it had been. Concerning Sewell's work, McIntyre states:

If the original observation of Sewell was correct, the curare used . . . seems to have differed considerably from those employed later; for, though convulsions can be interrupted by the curares recently used, convulsions may return when the curare concentration falls, whereas in Sewell's experiences the convulsions did not return after the animal had been resuscitated.

It is, perhaps, not surprising that McIntyre should have failed to find any evidence for the actual use of curare in hydrophobia, for the following lies buried in the body of an extensive article in the *Veterinarian*[8] for 1838. Youatt relates:

Mr. Morgan having kindly given me some of the Ticunas (arrow) poison, I inoculated a rabid dog with it, in order to see what effect one poison might have in weakening or destroying another. Although not ferocious, the animal had been in a considerable state of excitation. An incision through the integument was made on the inside of the arm, and a pointed bit of wood that had

been dipped in the poison was rubbed on the exposed fasciae. No effect being produced, the same bit of wood was introduced into an incision more deeply made. In less than two minutes the dog was more tranquil, and at the expiration of five minutes he dropped motionless, the only indication of his life being a regular and not laborious breathing. In this state he continued eight hours, when I left him for the night. On the following morning I found him dead.

In the following year, Youatt[9] records some of the interest in the use of curare, in which "the medical men of Nottingham, much to their credit, have taken the lead." He describes several experiments conducted before a group of medical men and veterinarians in which a dog and 2 asses were given curare; the dog was destroyed, both asses recovered, 1 after seven hours of artificial respiration. "The result of these experiments was exceedingly gratifying, and the gentlemen who conducted them resolved to avail themselves of the first opportunity to try them in tetanus or rabies." Later, a horse with an open joint ". . . was properly attended, but in the process of time, as might be expected (*sic*), tetanus appeared. It was immediately determined to have recourse to the wourali poison. Most of the medical gentlemen in Nottingham, and its neighbourhood, were present." A lengthy record, kept by one of the medical men in attendance, reveals that at the outset the jaws were firmly fixed and after four hours the patient attempted to eat, but later the symptoms reappeared in an aggravated form and the animal was destroyed.

DISCUSSION

Booth and Pierson[1] point out that, in the late nineteenth century, treatment of tetanus in horses with preparations of curare administered *per os* lost favor with veterinarians because of its lack of dependability. Whether parenteral administration, as in Sewell's cases, would have been any more dependable is a matter for conjecture but, with the advantage of hindsight, we can at least recognize a precedent for such a departure. In the same vein, while Booth and Pierson's frequent administration of d-tubocurarine undoubtedly achieved the peripheral effect for which they were looking, McIntyre[2] points out that the now well-known central effect of d-tubocurarine is apparently of greater duration and, under some circumstances, it is the primary effect. He says, "It seems probable that the results obtained clinically in spasticities and related conditions do not depend solely upon the classical peripheral actions . . . the central action of curares containing d-tubocurarine should be critically examined."

The primary purpose of this communication has been to bring to light this little-known facet of veterinary history. While it may be fruitless to speculate upon what might have been the turn of events had veterinarians been sufficiently aware of this information, this is but one of many similar cases.

With the advent of pure alkaloid curare compounds, it is apparent that the entire problem of the use of curare in the treatment of tetanus must be reinvestigated. In this, as in all investigations, as new information is added to the body of clinical data, we should keep in mind the warning of the eminent anesthesiologist, R. M. Waters,[10] that clinical practice (in anesthesia) is handicapped by failure to correlate and utilize existing knowledge, and "Permanent improvement will develop in the future, as it has come in the past, mainly through consideration of scientific facts—laborious learning, imaginative insight and accumulative application."

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Surgery and Obstetrics
and Problems of Breeding

Comparison of Some Local Anesthetics in Cattle

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IN RECENT YEARS, several new local anesthetics have been introduced. Those which have been adopted for clinical use have certain properties which warrant their use. Procaine, although it is widely used in veterinary medicine, is not a good topical anesthetic. When injected, its action is slow in onset and it is listed among the weaker local anesthetics.

In a comparison of some local anesthetics used in cattle,[13] cyclaine® (hexylcaine) produced a longer lasting effect than procaine. Additional reports[5,15] refer to the clinical effectiveness of cyclaine as a local anesthetic.

In a comparison of the pharmacological properties of xlyocaine® (lidocaine) and procaine,[8] xylocaine was found to produce the longer local anesthesia in laboratory animals. It has also been found to be superior to other local anesthetics used in animals.[1] Xylocaine has been reported to be less irritating and to have lower toxicity than cyclaine.[11] When the two drugs were compared for epidural anesthesia for abdominal surgery in man, it was found that both produced satisfactory anesthesia but there was greater incidence of systemic toxicity from xylocaine.[2] However, when biopsy sections of tissues infiltrated two days previously with the drugs were examined, no evidence of gross or microscopic tissue changes was found.[12] Tissue reaction to the antihistamines, pyribenzamine,® thenylene,® and antistine,® has been studied.[14] All three compounds produced regional and spinal anesthesia in rabbits but they had injurious effects on tissues. Pyribenzamine has been used as a local anesthetic in man.[3] The authors did not report any unusual tissue reaction.

This is a report of an investigation of the epidural, conduction (paravertebral), and infiltration anesthetic properties of cyclaine, xylocaine, procaine, and pyribenzamine. A minimum of eight tests on separate animals was made with each compound for each type of local anesthesia, and only one type was studied on an animal at one time. The latent period (interval between injection and onset of anesthesia), duration of anesthesia, and tissue reaction were determined for each anesthetic. Tissue reaction was studied by taking sections of the tissues six hours after the animals were injected.

METHODS

Mature dairy cattle weighing 850 to 1,400 lb. were used. None of the animals were affected with any detectable systemic infection, and no infection was evident in the areas where the infiltration type of anesthesia was studied. A 2 per cent solution of each anesthetic was used. Each solution also contained epinephrine, 1:100,000.

The cattle were confined in stocks or stanchions. The hair was clipped from, and tincture of iodine applied to, the skin where needles were to be introduced. The technique of injection for epidural anesthesia was that recommended by Benesch[4]; the method of injection for paravertebral anesthesia was that recommended by Farquharson.[7] An attempt was made to inject the same volume of anesthetic solution in approximately the same total area of tissue in the infiltration studies. Tissues in the paralumbar fossa were infiltrated.

The dose of anesthetic, injected epidurally, was calculated on the basis of approximately 1.25 ml./100 lb. of body weight. The solution was injected slowly. This dosage was calculated in an attempt to bring about satisfactory anesthesia without causing the animal to stagger or to be unable to stand. In the studies on paravertebral lumbar anesthesia, 7 ml. of anesthetic solution was injected in the vicinity of each nerve.

Duration of anesthesia was determined at intervals by pricking the skin with a needle or, in epidural anesthesia, by pinching the vulva with forceps and also by noting the response of the tail muscles. Good epidural anesthesia was indicated by paralysis of the tail, suspension of defecation, and lack of sensory reaction when the vulva was pinched. Complete absence of sensation in the area supplied by the nerves involved in the paralumbar block or in the tissues infiltrated was considered good anesthesia in the other phases of the study. Anesthesia was considered terminated when there was evidence of sensation in an area previously anesthetized.

From the Department of Veterinary Physiology and Pharmacology and the Agricultural Experiment Station, University of Illinois, Urbana. Dr. Smith is now at Kansas State College, Manhattan.

The assistance of Dr. A. M. Watrach is gratefully acknowledged by the authors.

The cyclaine used in this study was supplied through the courtesy of Dr. S. F. Scheidy, Sharp & Dohme Company, Philadelphia, Pa.

The xylocaine was supplied through the courtesy of Dr. G. Edds, Fort Dodge Laboratories, Fort Dodge, Iowa.

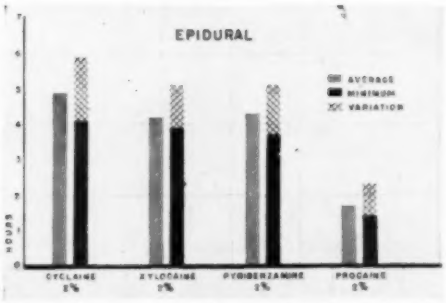

Fig. 1—The average, minimum, and maximum duration of anesthesia produced by four different compounds when injected epidurally in cattle.

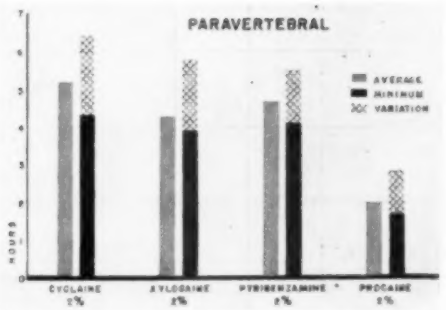

Fig. 2—The average, minimum, and maximum duration of anesthesia produced by four different compounds when used to block paravertebral nerves in cattle.

RESULTS

Epidural Anesthesia.—Cyclaine produced good anesthesia in 8 animals for an average of 4.9 hours. The latent period was short. Anesthesia developed in some animals within six minutes after injection. Three of the animals became unsteady but remained standing.

Xylocaine produced anesthesia similar to that observed when cyclaine was used except the latent period was shorter. The average duration of anesthesia in the 11 animals studied was also somewhat shorter —4.2 hours. Anesthesia appeared to be fully developed in 1 cow three minutes after the injection was completed.

Pyribenzamine produced good anesthesia in 10 of 11 animals; 1 showed no evidence of anesthesia. The average duration of anesthesia in the 10 animals was 4.3 hours. The latent period was slightly longer than with cyclaine.

Procaine produced good anesthesia in 8 of 9 animals, with an average duration of 1.7 hours. The latent period was the longest of the four compounds studied.

The average duration of epidural anesthesia and the minimum and maximum range are illustrated (fig. 1).

Paravertebral Anesthesia.—In general, the results from paravertebral injections (fig. 2) were similar to those obtained when the same compounds were injected epidurally. In the 9 animals studied, cyclaine produced good anesthesia which persisted an average of 5.2 hours.

Two animals injected with xylocaine did not develop satisfactory anesthesia but this may have been due to faulty technique. However, satisfactory paravertebral anesthesia was obtained in 8 of 10 animals, with the average duration of anesthesia 4.3 hours.

With pyribenzamine, the average duration of anesthesia in 8 animals was 4.7 hours. There was a narrow range of variation in the different animals and the latent period was short.

Procaine produced reasonably good anesthesia in 9 animals but the effect, as compared with the other three compounds, was not as profound or complete, the duration of anesthesia was much shorter (av. 2 hr.), and the latent period was longer.

Infiltration Anesthesia.—It is somewhat difficult to compare accurately different compounds as to their ability to produce and maintain anesthesia when infiltrated into tissues (fig. 3). However, an attempt was made to standardize the procedure. An effort was made to inject approximately the same volume of anesthetic solution per unit of tissue.

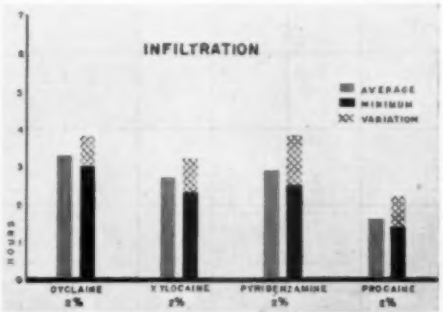

Fig. 3—The average, minimum, and maximum duration of local anesthesia produced when different compounds were infiltrated into tissues in the paralumbar fossae of cattle.

When cyclaine was infiltrated into the tissues of 9 animals, the average duration of anesthesia was 3.3 hours. Cyclaine appeared to have good diffusion properties and a short latent period.

Xylocaine produced good anesthesia when infiltrated into the tissues of 8 animals. The latent period was slightly shorter than with cyclaine, and xylocaine appeared to have the best diffusion properties of the four compounds tested. The average duration of anesthesia was 2.7 hours.

Pyribenzamine produced anesthesia which persisted 2.9 hours. It also had a short latent period and good diffusion properties.

With procaine, the anesthesia was slow in onset, of relatively short duration, and the solution did not appear to diffuse rapidly through the tissues. The average duration of anesthesia was 1.6 hours.

No evidence of systemic toxicity from the anesthetics was observed in any of the animals used in this study.

Tissue Reaction.—Histopathological examination of tissues taken six hours after injection revealed practically identical changes produced by the four compounds studied. There was a mild, acute inflammatory reaction in the muscle tissue characterized by emigration of polymorphonuclear leukocytes and a few mononuclear cells. These exudative changes were usually focal in distribution and appeared to be restricted to the loose connective tissue. No other changes of significance were observed in the tissues. Tissues injected with normal saline showed similar reactive phenomena. It would appear that the changes observed were of transitory and insignificant character.

DISCUSSION

The results reported in this study are similar to those reported by other workers. Xylocaine had the shortest latent period, followed by cyclaine, pyribenzamine, and procaine. It also appeared to have the best diffusion properties, although it did not appear to surpass cyclaine and pyribenzamine markedly in this characteristic. Cyclaine, as previously reported,[6] produced anesthesia of longest duration. Histological study of tissues injected with the different anesthetics did not indicate significant variation in irritant properties of the compounds, although xylocaine has been reported[10] to be less irritating than cyclaine. Evidence of systemic toxicity was not observed in any of the animals used in this study. However, others[2] have concluded that xylocaine produced symptoms of toxicity in a higher percentage of human patients than did cyclaine.

It has been observed that when 20 ml. of 1 per cent xylocaine was injected into a cow through a caudal catheter, it produced surgical anesthesia for 59 minutes,[9] and subsequent injection of 1 per cent cyclaine produced surgical anesthesia for only 38 minutes. These periods of anesthesia were much shorter than those obtained in our study. The size of the cow and whether epinephrine was combined with the anesthetics was not indicated.

Since tissue reaction was studied at only one time interval after the anesthetics were injected, it was not possible to evaluate the duration of hyperemic activity produced. It has been reported[10] that the long-lasting hyperemic activity of xylocaine is of special value in treatment of arthropathic conditions.

Whether failure to obtain anesthesia in some cases following the injection of a local anesthetic is always due to faulty technique is unknown. However, Hingson,[9] who has administered local anesthetics to thousands of human patients, reports: "We have been intrigued (in our prolonged continuous conduction anesthesias) that a given patient who has on occasion had two or three times the average adequate conduction anesthesia in the extradural or subarachnoid space has failed to obtain anesthesia. Even though such a phenomenon may on occasion be due to inaccurate positioning of the needle, we have proved that they occur just as frequently in the presence of perfect technique. In such cases the mere substitution of another anesthetic agent has produced prompt and adequate anesthesia."

SUMMARY

Two per cent solutions of cyclaine,® procaine, pyribenzamine,® and xylocaine® containing 1:100,000 epinephrine were compared as to duration of epidural, paravertebral, and local infiltration anesthesia. Latent period and tissue reaction for each compound were also recorded.

Cyclaine produced anesthesia of longest duration, followed in order by pyribenzamine, xylocaine, and procaine, duration with procaine being much shorter than with the others. Xylocaine, which appeared

to have the shortest latent period and the best diffusion properties, did not markedly surpass cyclaine and pyribenzamine in these properties. Procaine had a relatively long latent period and fair diffusion properties.

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Gallstones Obstruct Intestine.—Intestinal obstruction in man due to gallstones has been reported in 25 cases from one clinic. Some were relieved surgically, others recovered spontaneously, with the cholelith being recovered after passage. Autopsy in one person revealed a cholelith, 3.5 by 2.5 by 3.0 cm. lodged 3 ft. proximal to the ileocecal valve.—*J.Am.M.A., July 7, 1956.*

The Occurrence of So-Called "White Heifer Disease" in a White Shetland Pony Mare

CARL F. SCHLOTTHAUER, D.V.M., and
PAUL E. ZOLLMAN, D.V.M.

Rochester, Minnesota

The condition known as "white heifer disease" is a gross genital abnormality occurring most frequently in white Shorthorn heifers but, occasionally, in roan and in red and white heifers. It has been recognized clinically for many years by veterinarians in Great Britain. Williams,[1] in America, however, does not mention it in his book and we did not find any mention of it in American veterinary literature.

Spriggs,[2] in discussing white heifer disease, mentioned that he had found no single lesion that was characteristic of this condition but that certain lesions occurred more frequently than others, namely: (1) imperforate hymen, the vagina often terminating at this point; (2) cystic distention of the uterine horns, the uterine body remaining rudimentary; and (3) complete absence of the cervix and anterior portion of the vagina.

White heifer disease undoubtedly has been observed in heifers by veterinarians in America, but was reported as a congenital abnormality. The following case is peculiar only in that it occurred in a white Shetland pony mare.

CASE REPORT

A white, female Shetland pony, 2 years old, was brought to us for diagnosis. The owner stated that the pony had had a purulent vaginal discharge for about a year. He had treated the condition with antiseptic vaginal douches, various sulfonamides given by mouth, and penicillin by intramuscular injection, with only temporary benefits, if any. When first seen by us, pus and urine seemed to dribble almost constantly through the vulva. The vulva was normal in size, but a necrotic pseudomembrane on its inner margin seemed to extend into the vagina. The medial surfaces of the thighs and the hocks were denuded, ulcerated, and bleeding, due to the dribbling of urine and pus. After cleansing and

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treating the visible lesions, the pony was given 2,000,000 units of penicillin intramuscularly in an attempt to control some of the infection and to make further examination possible. Marked improvement was observed within 48 hours, but it was of short duration. Because of the ulceration of the vulva, an examination of the vagina was not attempted. A congenital anomaly was suspected but, because of the size of the pony, rectal palpation of the genital tract was impossible. Euthanasia was advised.

Necropsy revealed that the vagina was present but the cervix was absent. The uterus was atrophic and had no outlet into the vagina. The right uterine horn was 14.0 cm. long by 4.0 to 6.0 cm. in diameter and contained clear fluid; the left horn was 5.0 cm. long and 0.3 cm. in diameter. The bladder was absent. The right kidney measured 10.0 by 10.0 by 8.0 cm. and the ureter entered the anterolateral portion of the vagina. The left kidney was atrophic —4.5 cm. long, 2.5 cm. wide, and 0.6 cm. thick and the ureter was absent.

Apparently so-called white heifer disease can also occur in the mare.

References

[1]Williams, W. L.: Diseases of the Genital Organs of Domestic Animals. Privately published. Ithaca, N. Y., 1921.

[2]Spriggs, D. N.: White Heifer Disease. Vet. Rec., 58, (1946): 405-409, 415-418.

Intestinal Anastomosis and Repair of Inguinal Hernia in a Dog Under Septic Conditions

HOWARD O. WEBER, D.V.M.

Simsbury, Connecticut

On Feb. 24, 1956, a 6-year-old black, male Cocker Spaniel was brought for treatment following an automobile accident. Apparently only suffering from bruises, it was discharged on February 26, showing a slight stiffness in the posterior parts. On February 28, the dog was brought back with a painful swelling in the right inguinal region which was diagnosed as an inguinal hernia.

The dog was then prepared for surgery and anesthetized with pentobarbital sodium. An incision was made through the skin and subcutaneous tissues, and a gangrenous loop of intestine, 6 inches long and 1 inch in diameter, was found in the hernial sac. The intestine ruptured when an attempt was made to free it and the contents spilled into the sac. In spite of the gangrene, the condition of the animal was excellent and it was decided to complete the operation.

The area was cleansed as well as possible with phisohex, the hernia ring was enlarged, the strangulated intestine was drawn out to where the bowel appeared healthy, and the 6 inches of gangrenous intestine was excised. Anastomosis was accomplished using Cushing sutures with No. 0 intestinal gut. The intestine was then replaced in the abdominal cavity, followed with liberal amounts of antiseptic powder.* The inguinal canal was then closed, using No. 1 chromic gut and the skin was sutured with No. 2 silk.

Following the operation, 250 cc. of a 5 per cent dextrose and saline solution was given intravenously and 300,000 units of penicillin was given intramuscularly and was repeated daily. The dog was returned to a cage and kept warm until he recovered from the anesthetic.

The following day, the dog drank a little milk and for five days he ate horsemeat and cereal gruel readily. The third day following the operation, the skin wound disrupted, undoubtedly due to contamination, leaving a 4-inch opening. This was left without resuturing.

The dog was discharged on March 15 in good spirits. The owner reported that the wound finally healed by May 1, 1956, and that the dog seemed normal.

The conclusion reached by the author, from this operation, is that certain dogs can withstand an enormous amount of contamination of the abdominal cavity when sufficient antibiotics are given.

Dr. Weber is a practitioner in Simsbury Conn.

*Furacin soluble powder, produced by Eaton Laboratories, Norwich, N. Y.

Estrogens to Terminate Lactation.—Ethinyl estradiol was given to 27 women who had just weaned and to three who were still nursing babies. In all 30, the flow of milk ceased completely in five to 13 days after starting therapy. The total dose required was from 1.2 to 1.9 mg. The estrogen probably acts directly on the hypophysis by inhibiting the production of prolactin or luteotropin.—*J.Am.M.A., May 26, 1956.*

Polioencephalomalacia of Cattle and Sheep

RUE JENSEN, D.V.M., Ph.D.; L. A. GRINER, M.S., D.V.M.; O. R. ADAMS, D.V.M., M.S.

Fort Collins, Colorado

POLIOENCEPHALOMALACIA, a noninfectious disease of pasture and feedlot cattle and sheep, is characterized by multiple foci of necrosis in the cerebral cortex. In Colorado, the disease is known as "forage poisoning." In Wyoming, where the disease has been studied extensively, it is known as "blind staggers" from selenium poisoning. The clinical syndromes of the disease in Colorado and Wyoming are identical. The cause of the disease in cattle and sheep of Colorado has not been studied adequately, while the neuropathology of the disease in cattle and sheep of Wyoming has not been reported.

Although it is assumed that blind staggers reported from Wyoming, and forage poisoning reported from Colorado, are a single entity, the appellation polioencephalomalacia is appropriate until the etiological and pathological factors are clearly established. This paper is primarily a presentation of the neuropathology of polioencephalomalacia.

REVIEW OF LITERATURE

In studies in Wyoming, selenium poisoning in cattle was classified[1,2] as occurring in two forms: acute or blind staggers, and chronic or alkali disease. The signs of the acute form were progressive impairment of vision, wandering from the herd, circling movements, depraved appetite, paralysis, abdominal pain, and terminal respiratory failure. The signs of the chronic form included emaciation, stiffness, lameness, mental dullness, swelling at the coronary band, separation of the hoofs from the skin, deformity of the hoofs, and loss of hair from the switch of the tail.

Approximately 100 necropsies were performed on cattle and sheep affected with selenium poisoning. The disease was subdivided into two forms: blind staggers and alkali disease. For the chronic or blind staggers form, the gross pathological changes included congestion and focal necrosis of the liver, congestion of the kidneys, petechiae on the heart with endocarditis and myocarditis, excessive fluid in the pericardial sac, and erosion of the proximal articular surfaces of the metatarsi. The histopathological changes included cloudy swelling, necrosis, and fibrosis of the liver; periangitis, serofibrinous myocarditis, and hemorrhages of the heart; and glomerulonephritis. For the acute or alkali form of the disease, the gross pathological changes were cirrhosis of the liver; endocarditis, myocarditis, and hemorrhages of the heart; hemorrhages in the medulla of the kidney; elongation, deformity and fissuring of the hoofs; and gelatinous degeneration of bone marrow. The histopathological changes were cirrhosis and atrophy of the liver; myocarditis and endocarditis; and glomerulonephritis, intertubular hemorrhage, and hyalinization of convoluted tubules of the kidney.

Later the pathological changes of selenium poisoning were studied[3] in both naturally and experimentally affected cattle and sheep. The disease was then classified as either acute, subacute, or chronic. The chronic type was further subdivided into two forms: blind staggers and alkali disease.

In the acute form, gross pathological changes included serosal hemorrhages; petechiation and enlargement of the heart; edema and hemorrhages of the lungs; softening, enlargement, and necrosis of the liver; and softening and swelling of the kidneys. Histopathological changes included necrosis, hemorrhages, edema, and congestion of abdominal and thoracic viscera.

In the subacute form, gross pathological changes were firmness of the heart, fibrosis of the lungs, firmness and granularity of the liver, and atrophy and fibrosis of the kidneys. The histopathological changes were fibrosis of the heart and lungs, and fibrosis and bile duct proliferation in the liver.

In the blind staggers type of the chronic form, gross pathological changes were hemorrhages and atrophy of the heart, congestion and fibrosis of the lungs, and atrophy of liver and kidneys. The histopathological changes were periangitis and fibrosis of the heart; fatty metamorphosis, necrosis, and fibrosis of the liver; parenchymatous degeneration, intertubular hemorrhage, hyaline degeneration, glomerulonephritis, and fibrosis of the kidneys.

In the alkali disease type of the chronic form, gross pathological changes included softness, atrophy, and fibrosis of the heart; congestion and fibrosis of the lungs; atrophy and fibrosis of the liver; fibrosis of the kidneys; and ascites.

Further investigation[4] confirmed the above classification and extranervous pathological findings of selenium poisoning but, in addition, found congestion, hemorrhage, edema, and softening of the brain.

A review of the general subject of selenium poisoning[5] resulted in concurrence in the above classification of the disease and in the signs manifested by the different types.

The minimum lethal oral dose of selenium for cattle was found to be 4.5 to 5.0 mg. per pound of body weight.[3] The signs shown by experimentally poisoned cattle were anorexia, general depres-

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sion, acceleration of pulse and respirations, dilation of nostrils, and normal or slight elevation of body temperature.

OCCURRENCE

Cattle.—Polioencephalomalacia has been diagnosed in cattle of Colorado, Wyoming, western Nebraska, and western Kansas.

TABLE 1—Estimated Annual Incidence of Bovine Polioencephalomalacia in Four Areas of Colorado*

Area	Number		Cases per 10,000 cattle
	Cattle	Cases	
Fort Collins	70,000	125	17.9
Greeley	240,000	235	9.8
Lamar	100,000	1,000	100.0
Longmont	150,000	65	4.3

*Estimates were obtained from Dr. S. W. Beggs, Lamar; Weld County Veterinary Medical Association, Greeley; Drs. J. W. Harrison and R. Hargreaves, Longmont; and the Veterinary Hospital, Colorado A. & M. College, Fort Collins.

The estimated annual incidence for four areas in Colorado is shown (table 1).

In a series of 111 fatal cases, 42 (38%) were males and 69 (62%) were females. The cattle population for the area was predominantly female.

The incidence by age in 110 cases (table 2) showed the larger number in the 12- to 18-month age group.

The incidence by breed in 107 cases (table 3) showed Hereford cattle, which predominated in pastures and feedlots, with the higher incidence.

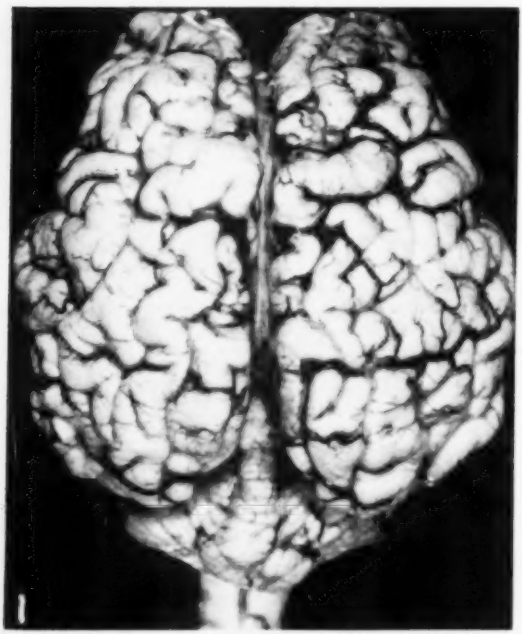


Fig. 1—Brain of normal cow, dorsal view. x 0.8.

TABLE 2—Incidence by Age of 110 Cases of Bovine Polioencephalomalacia

Months	1-6	7-12	13-18	19-24	25-36	Older
Cases	8	23	56	4	6	13

TABLE 3—Incidence by Breed of 107 Cases of Bovine Polioencephalomalacia

Breed	Hereford	Angus	Holstein	Shorthorn	Guernsey
Cases	71	20	13	3	1

TABLE 4—Monthly Incidence of 103 Cases of Polioencephalomalacia in Feedlot and Pasture Cattle

Month	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
Feedlot	22	7	—	2	2	1	5	1	2	2	1	1	44
Pasture	—	—	1	—	1	3	29	14	8	1	1	1	59

The monthly incidence of polioencephalomalacia in 103 cases in cattle from feedlots and pastures (table 4) showed a higher incidence for feedlot cattle during January, and for pastured cattle during July. On pasture, the disease commonly developed five to ten days following change from overgrazed to good pasture.

Sheep.—Although the disease was not common in sheep, polioencephalomalacia has been observed in two flocks (table 5).

TABLE 5—Polioencephalomalacia in Two Flocks of Sheep

Flock	Management	Number		Month
		Sheep	Cases	
A	feedlot	1,200	70	Jan.
B	pasture	90	7	Aug.

ETIOLOGY

Research workers in Wyoming are of the opinion that blind staggers in cattle in that state is caused by chronic selenium poisoning.[1,2,5,6]

In Colorado, the cause of forage poisoning has not been determined. Analyses of organs from affected cattle have shown a wide range of concentration of selenium. Attempts have been made, unsuccessfully, to reproduce the disease experimentally by forcibly feeding healthy cattle with stomach and intestinal contents from cattle in early stages of the disease, by parenteral inoculation of experimental cattle with blood and ground parenchymatous organs from animals in early stages of the disease, and by forcibly feeding cattle with large amounts

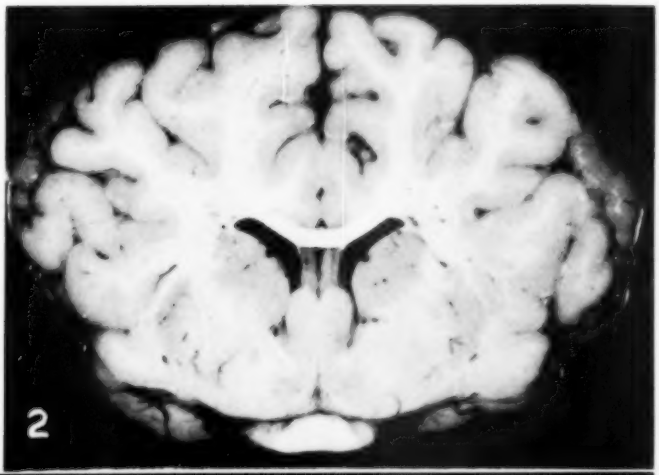


Fig. 2—Cross section of brain from normal cow. x 1.0.

Fig. 3—Cross section of brain from a cow affected with acute polioencephalomalacia. (a) Foci of necrosis in cerebral cortex. x 0.9.

Fig. 4—Cross section of brain from a steer affected with acute polioencephalomalacia. Necrotic foci (a) and separation of necrotic cortex (b) from white matter. x 0.7.

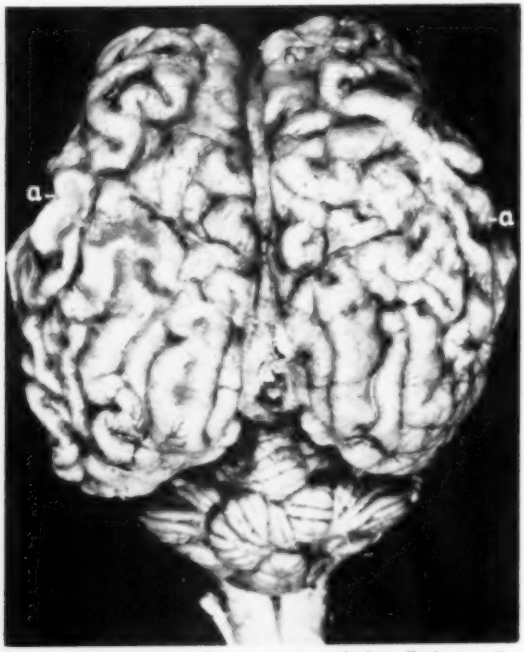


Fig. 5—Brain of a cow recovered clinically from polioencephalomalacia. Necrotic cortex has been removed at (a). x 0.73.

of water and plants from areas where the disease developed. Feeding of suspected materials was for short periods only. The study of toxins in the intestines of acutely affected cattle and of antitoxins in the plasma of recovered cattle was inconclusive.

SYMPTOMATOLOGY

Two degrees of severity of forage poisoning were recognized clinically. The more severe type was usually seen in feedlot animals and only occasionally in the pastured animal. Animals affected with the acute type were often found prostrate and comatose. During early stages, rarely seen, the animals suddenly backed away from the feed bunk, showed severe muscular tremors, twitching of the ears, eyelids, and facial muscles, and occasionally convulsions. Bilateral impairment of vision was constant. No pathological changes of the eye were discernible. The pupils reacted normally or slowly to light. The visible mucous membranes were injected. The pulse and respiration rates were accelerated, especially during convulsions when the temperatures were above normal.

Animals affected with the mild or subacute form were usually found in summer pastures. They separated from the herd

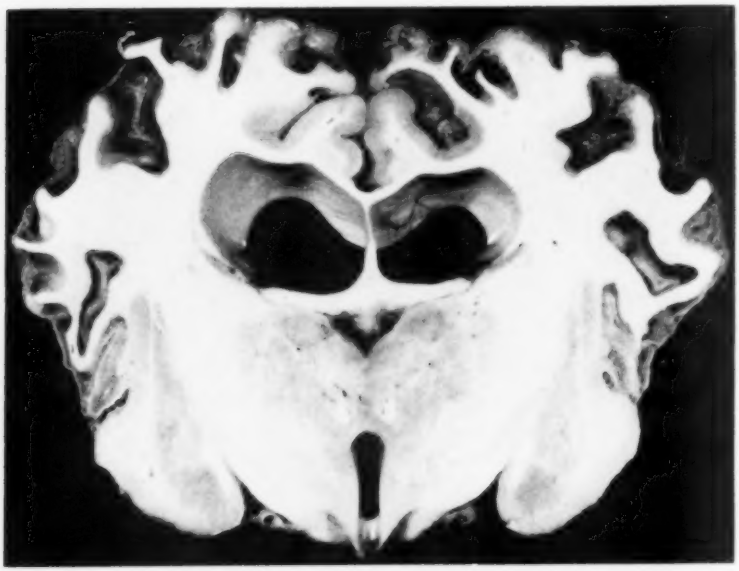


Fig. 6—Cross section of brain from a cow recovered clinically from polioencephalomalacia. Nearly all cerebral cortex at this plane has been necrosed and removed. x 1.0.

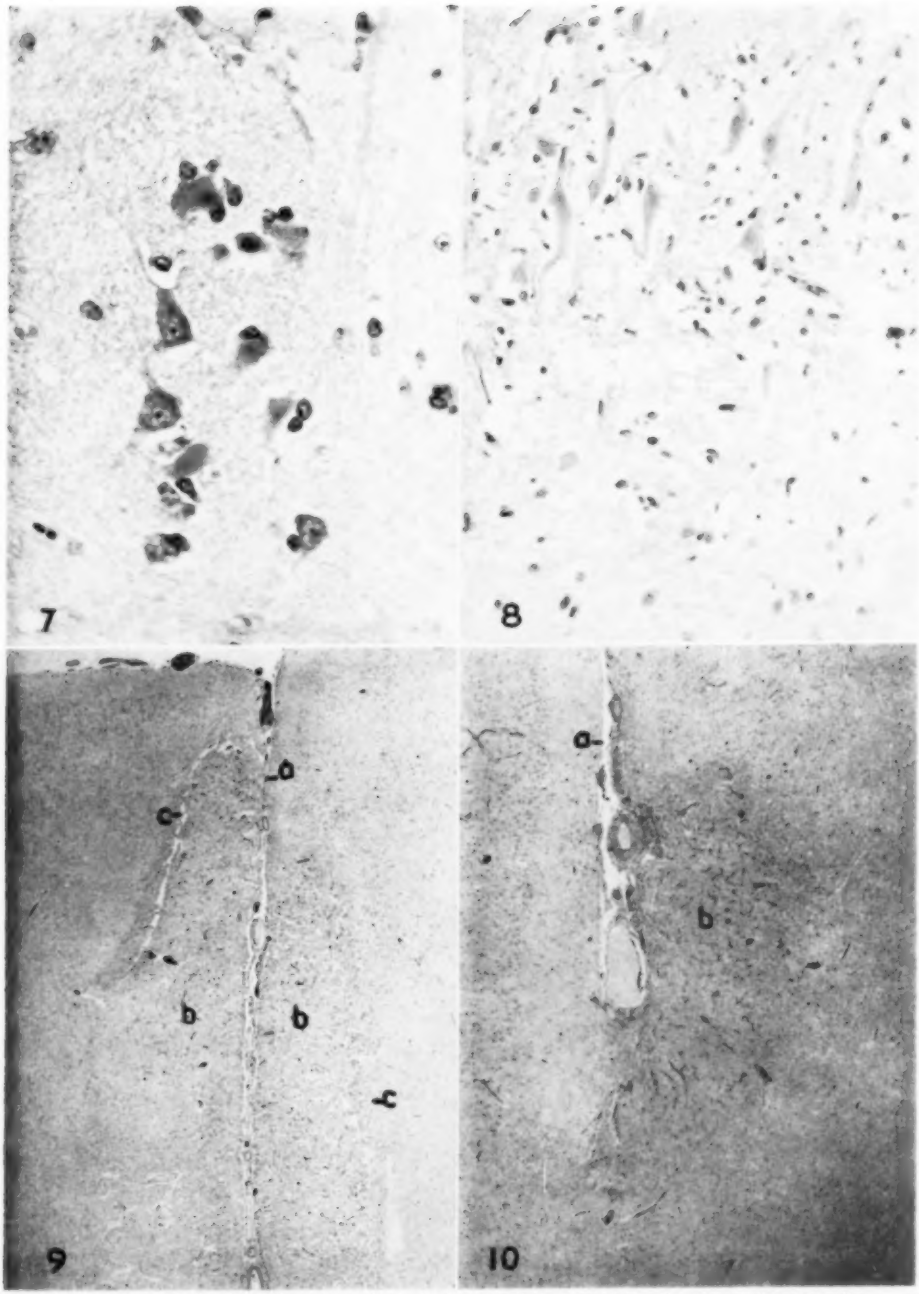


Fig. 7—Neurons of ectosylvian sulcus of cerebral cortex from a cow in early stages of acute polioencephalomalacia. The section was from an area at junction of viable and necrotic tissue. The two neurons at left center are viable; other neurons are necrotic. Nocht's stain. x 570.

Fig. 8—Cerebral cortex at coronal sulcus from a cow in early stages of acute polioencephalomalacia. All neurons are necrotic. Nocht's stain. x 160.

Fig. 9—Lateral sulcus of cerebral cortex of a steer affected with acute polioencephalmalacia—(a) sulcus, (b) necrotic tissue, and (c) line of separation from viable tissue. Nocht's stain. x 12.

Fig. 10—Cerebral cortex at callosomarginal sulcus (a) from a steer with acute polioencephalomalacia. (b) Necrotic tissue. Nocht's stain. x 22.

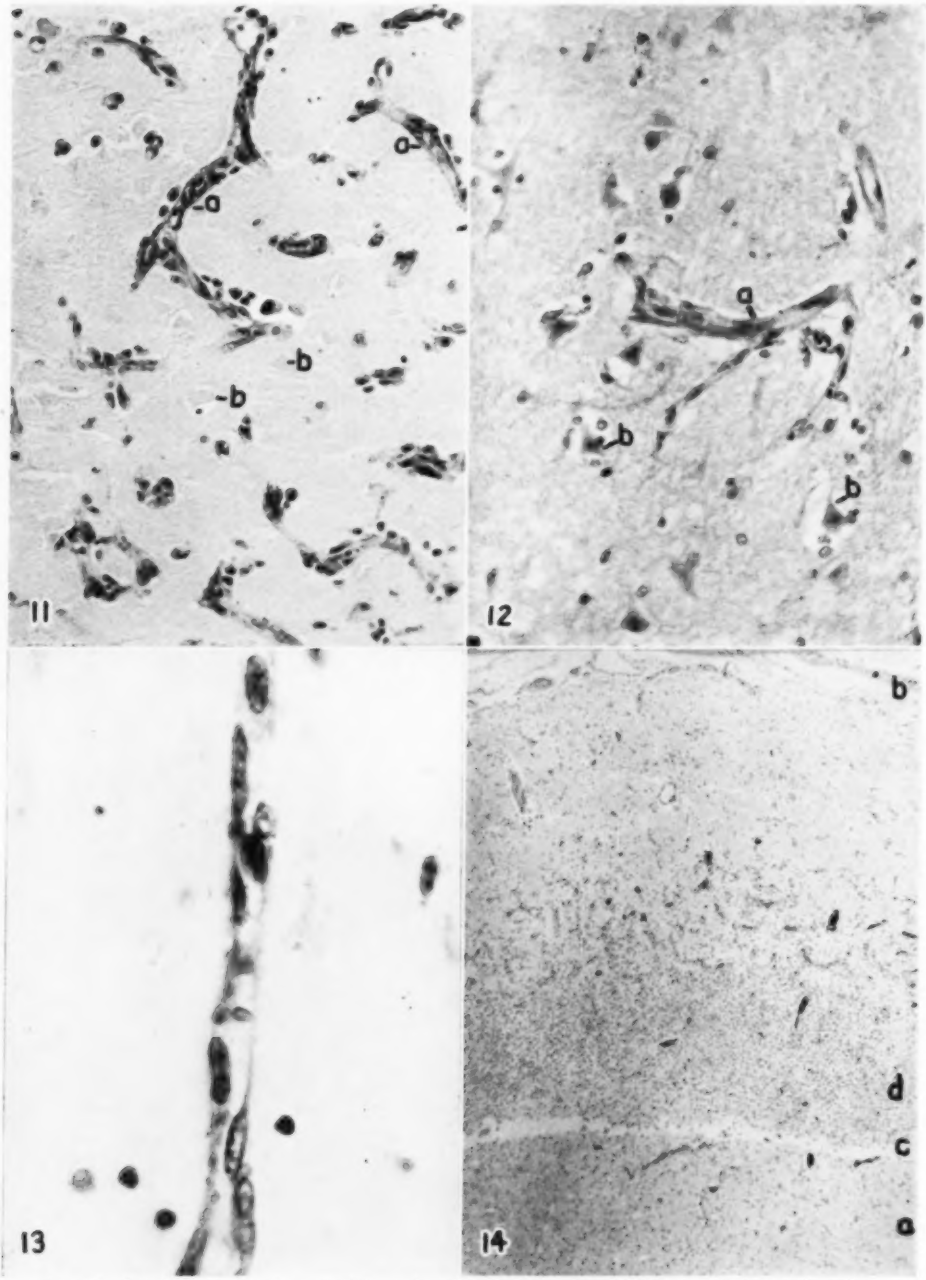


Fig. 11—Cerebral cortex at suprasylvian sulcus from a cow affected for several days with polioencephalomalacia—(a) proliferated capillaries and (b) necrotic neurons. Nocht's stain. x 150.

Fig. 12—Necrotic cerebral cortex at ectosylvian sulcus from a cow affected for several days with polioencephalomalacia—(a) proliferating endothelial cells; (b) necrotic neurons. Nocht's stain. x 320.

Fig. 13—A capillary with proliferating endothelial cells in necrotic coronal sulcus of cerebral cortex. Nocht's stain. x 645.

Fig. 14—Necrosis in calcarine sulcus of cerebral cortex of a cow affected several days with acute polioencephalomalacia—(a) viable tissue, (b) meninges, (c) line of separation of necrotic tissue, and (d) compound granular cells. Tissue from (a) to (d) is necrotic. Nocht's stain. x 31.

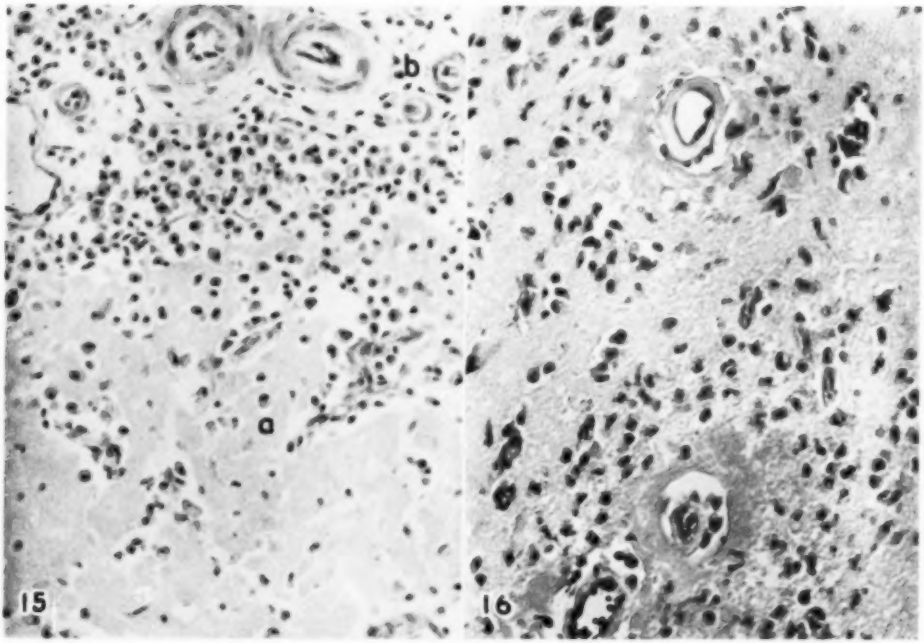


Fig. 15—Compound granular cells in area of necrosis in calcarine sulcus of cerebral cortex (a) and in adjacent meninges (b) from a steer affected for several days with acute polioencephalomalacia. Nocht's stain. x 177.

Fig. 16—Necrosis and infiltration of compound granular cells in perivascular tissue of suprasylvian sulcus of cerebral cortex from a cow affected several days with acute polioencephalmalacia. Nocht's stain. x 200.

and occasionally pushed against solid objects. They were bilaterally blind but with no visible changes in the eye. Twitching of muscles of the face or ears was present in some cases. Empty masticatory movements, excessive salivation, and bulbar paralysis were observed. The mucous membranes presented varying degrees of congestion. Temperatures remained near normal except when complications occurred. The pulse and respiration rates were normal or slightly increased. Depression was severe and response to stimulation was slow. Prehension was impaired in some animals, but they were able to masticate feed placed in the mouth; it permanently failed in others, which was fatal.

The course was one to several days. Recovery was complete or associated with permanent mental dullness and impaired vision. Case mortality was approximately 90 per cent for the severe form and 50 per cent for the mild form.

PATHOLOGY

The pathological changes of polioencephalomalacia were determined from necropsy of 105 cattle and 18 sheep. Of the 105 cattle, 98 were in acute stages and 7 had recovered clinically one to five months previously. Of the 18 sheep, 17 were in acute stages and 1 had recovered clinically. All viscera were examined carefully.

The brain, spinal cord, and samples of other viscera were fixed in 4 per cent aqueous formaldehyde. Following fixation, each brain was subdivided into transverse sections each approximately 1 cm. in thickness. The sections were examined for gross lesions. Paraffin sections were prepared from the following areas of the brain: coronal, suprasylvian, callosomarginal, lateral, rhinal, calcarine and ectosylvian sulci and adjacent gyri, insula, thalamus, hypothalamus, amygdaloid nucleus, corpus striatum, midbrain, pons, medulla, middle cerebellar peduncle, cerebellum, and spinal cord. Sections were stained with hematoxylin and eosin and by the methods of Van Giesen, Nocht, Weil, and Holtzer.

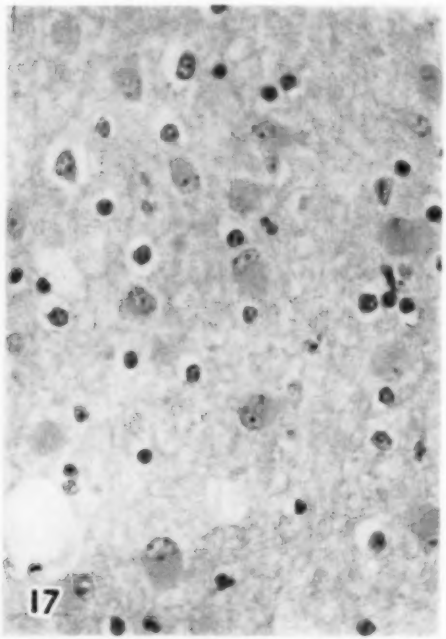


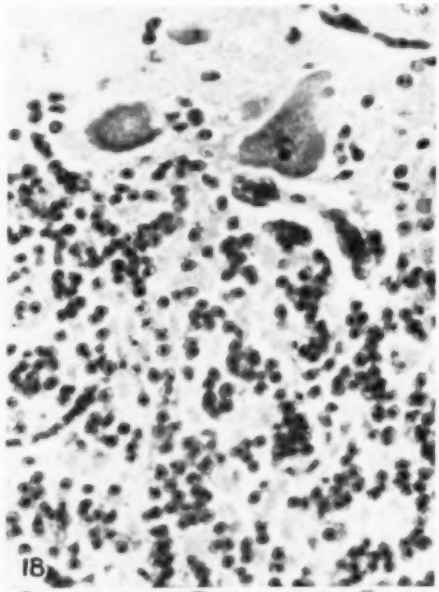
Fig. 17—Gemastete astrocytes in ectosylvian sulcus of cerebral cortex near an area of necrosis from a cow affected for several days with acute polioencephalomalacia. Nocht's stain. x 320.

GROSS PATHOLOGY

Acute Form.—Fresh or unfixed brains were excessively soft in affected areas of the cerebral cortex. Gross sections showed multiple foci of necrosis throughout the cerebral cortex, especially in the coronal, suprasylvian, lateral, calcarine, and ectosylvian sulci and adjacent gyri. Most foci possessed a slight yellowish discoloration. Frequently a line of cleavage between necrotic and viable tissue was discernible. Of the 98 brains from cows with acute cases, 69 contained discernible lesions in the cerebral cortex and 6 contained hemorrhages in the thalami in addition to cortical necrosis. Brains from normal cattle (fig. 1, 2), as well as brains from cattle affected with acute polioencephalomalacia (fig. 3, 4), are shown.

Extracephalic lesions included subendocardial and subepicardial petechiae and cloudy swelling in the liver.

Clinically Recovered Form.—Lesions in animals which had recovered were limited to the brains which were abnormally small. The intact brains and gross sections pre-



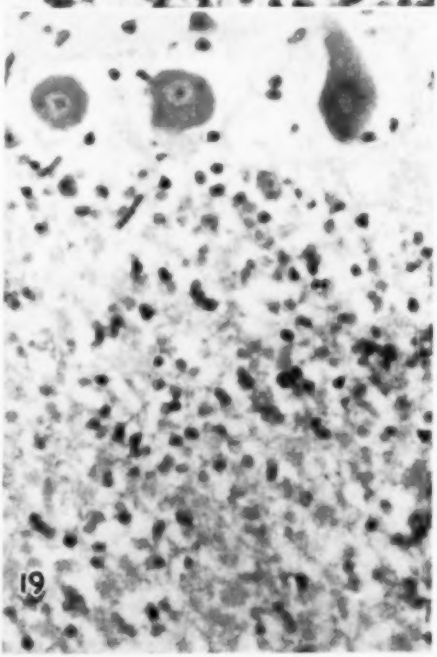


Fig. 18—Granular cells (below) and Purkinje cells (above) from cortex of cerebellum of a normal cow. Nocht's stain. x 320.

Fig. 19—Cerebellar cortex from a cow affected with acute polioencephalomalacia. Purkinje cells are above; necrotic granular cells, below. Nocht's stain. x 320.

sented foci of decortication where the meninges were in contact with white matter and where sulci were excessively wide. Small cysts were also present in the cerebral cortex. One cyst was found in a thalamus. Brains with loss of considerable cerebral cortex also contained compensatory internal hydrocephalus. Intact and cut surfaces of a brain from a cow which recovered clinically are shown (fig. 5, 6).

HISTOPATHOLOGY

Acute Form.—The sulci studied were affected constantly with necrosis. The size of necrotic foci varied up to several millimeters in diameter. In necrotic foci, either the entire thickness or part of the cortex was affected. In no case did necrosis extend into white matter. In the cerebral cortex from animals which had been sick one to two days, the neurons of affected areas were necrotic. Nuclei showed pyknosis, karyorrhexis, or chromatolysis, while the cytoplasm presented homogeneous eosinophilia with occasional vacuolization. Pericellular edema was common. At the margin of the necrotic area, both necrotic and viable neurons were present. Neuroglial cells were not affected to the same degree as were the neurons. Small hemorrhages were common. Some arterioles and capillaries showed swelling of endothelial cells.

In brains from animals which had been sick three days or more, the necrotic neurons were contracted into globular eosinophilic masses.

At the zone of junction of viable and necrotic tissue, compound granular cells containing phagocytized fat and tissue debris had accumulated and the dead tissue was being liquefied. In some areas, dead tissue was partly separated from viable tissue. Rod cells and gemästete astrocytes were discernible in viable tissue adjacent to necrosis. In viable tissue immediately surrounding dead tissue, astrocytes were increased in number. Endothelial cells proliferated to increase cellularity in small arterioles and to form new capillaries in the dead tissue and adjacent viable tissue. In adjacent viable tissue, some spaces of Virchow-Robin contained plasma protein and minimal numbers of lymphocytes and plasma cells. The leptomeninges over necrotic areas, which included the molecular layer, were infiltrated with compound granular cells and few lymphocytes and plasma cells. Cerebral histopathological changes of the acute form are shown (fig. 7-17).

Of the 98 brains from cattle in acute phases of the disease, 20 showed variable amounts of necrosis in the granular layer of the cerebellum.

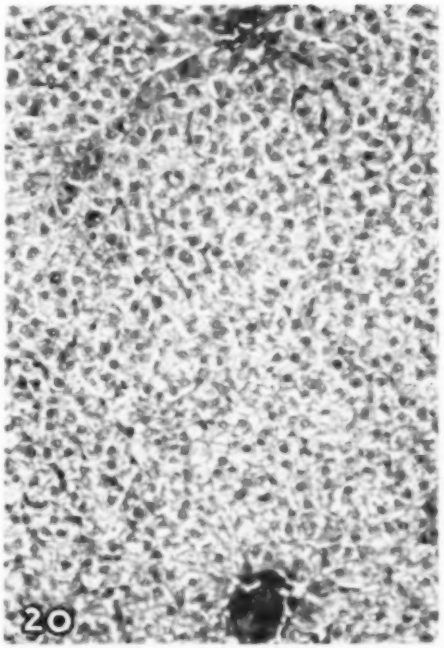


Fig. 20—Cloudy swelling in liver from a steer affected with acute polioencephalomalacia. Hematoxylin and eosin stain. x 160.

In case of severe changes, the granular cells were reduced in number and persisting necrotic cells showed pyknosis and chromatolysis. The histology of normal bovine cerebellum (fig. 18), and the histopathology of the cerebellum (fig. 19) and liver (fig. 20) from a cow with acute polioencephalomalacia are presented.

Extracortical and extracerebellar areas of the brain presented few isolated necrotic neurons. Thalami often showed hemorrhage.

Clinically Recovered Form.—Pathological changes were limited to the brain and almost exclusively to the cerebral cortex. In affected cortical foci, all dead tissue had been removed. Usually cysts were present.

In areas where necrosis had affected all layers of the cortex, the leptomeninges and white matter were in contact or were separated by an empty space. The cavities of cysts contained abundant nonfunctional capillaries. The viable tissue surrounding the cystic spaces was composed of astrocytes and astrocytic fibrils. The histopathology of clinically recovered cases of polioencephalomalacia are shown (fig. 21, 22).

DISCUSSION

Both acute and chronic selenium poisoning have been described in cattle.[1-6] Chronic

poisoning is characterized by hepatic injury, loss of hair from the switch of the tail, and deformity and lengthening of the hoofs. In Colorado, chronic selenium poisoning has rarely been seen and never in cattle which recovered from acute polioencephalomalacia. The absence of manifestation of chronic poisoning, in a cattle population where acute polioencephalomalacia is common, is irreconcilable with the theory that polioencephalomalacia is caused by acute selenium poisoning.

The minimal inflammatory reaction in meninges and viable nerve tissue adjacent to foci of necrosis is interpreted as being secondary to the necrosis.

Impairment of vision in polioencephalomalacia probably was caused by cortical injury in the region of the calcarine fissure. Permanent blindness resulted from necrosis of neurons, while recovery of vision followed nonlethal injury to neurons.

Primary and prominent lesions of polioencephalomalacia were limited to the brain. Extranervous lesions, hemorrhages, and cloudy swelling were nonspecific and of no diagnostic value.

Treatment

Treatment was supportive and consisted of purgation and administration of fluids and dextrose intravenously, and feed and stimulants orally.

In affected herds, additional cases were prevented by changing feed or pasture.

Summary

Polioencephalomalacia, a noncontagious disease of cattle and sheep, is characterized by focal necrosis disseminated throughout the cerebral cortex. The disease occurs in both feedlot and pastured cattle and sheep, and is known in Wyoming as blind staggers and in Colorado as forage poisoning. Acute and clinically recovered forms are recognized.

Necropsies on 105 cattle and 18 sheep revealed that primary significant lesions were limited to the brain.

Gross neuropathological changes in the acute form included multiple yellowish foci of necrosis in the cerebral cortex, beginning separation of affected tissue, and softness. In clinically recovered animals, gross

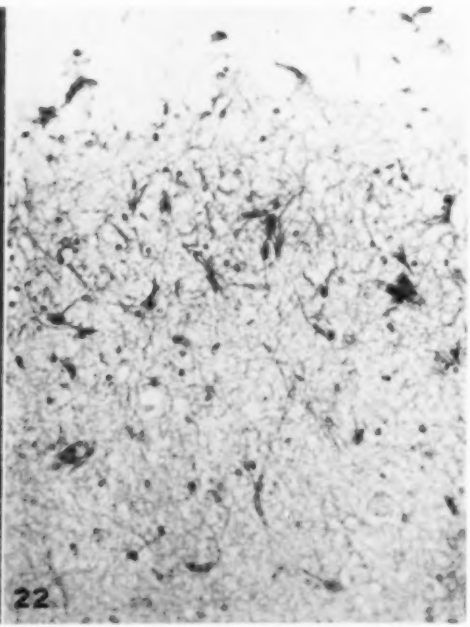


Fig. 21—Cavity in cerebral cortex at coronal sulcus from a cow recovered clinically from polioencephalomalacia. Lysis and removal of necrotic tissue created the cavity. Weil's stain x 15.

Fig. 22—Astrocytes and astrocytic fibrils in wall surrounding cavity in coronal sulcus of cerebral cortex from a cow recovered clinically from polioencephalomalacia. Holtzer stain. x 150.

lesions were cortical cysts and empty spaces from removal of necrotic tissue.

Neurohistopathological changes in acute cases included necrosis of neurons, proliferation of endothelial cells, accumulation of compound granular cells, and gliosis at the junction of necrotic and viable tissue. In clinically recovered animals, empty spaces and cysts in the cerebral cortex were surrounded by gliosis.

Although the cause of polioencephalomalacia is not positively known, clinical and pathological manifestations suggest intoxication.

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[3]Miller, W. T., and Williams, K. T.: Minimum Lethal Dose of Selenium, as Sodium Selenite, for Horses, Mules, Cattle, and Swine. J. Agric. Res., *60*, (1940): 163.

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Infectious Keratoconjunctivitis.—Rickettsia-like organisms were demonstrated in smears from the conjunctiva and in fluid from the anterior chamber of affected cattle; also in incubated eggs inoculated with bacteria-free fluid from the anterior chamber. After intraocular infection with egg cultures, 2 healthy calves developed typical symptoms and the organisms were again demonstrated.—*Vet. Bull., July, 1956.*

Effect of Pasteurization on Penicillin in Milk.—The effect of pasteurization on penicillin in milk was determined by adding five types of the antibiotic to samples of milk which were heated variously. At 143 F. for 30 minutes, the antibiotic potency loss was smallest in potassium penicillin 0 (0.9%), next in procaine penicillin (7.6%), penicillin K (10.9%), penicillin G salt (13.1%), and greatest in ephenamine penicillin G (16.1%). The loss of activity in penicillin K increased as follows: in milk, at 160 F. for 30 minutes (10.1%) and at 250 F. for 15 minutes (59.7%); and in water the loss was half at 143 F. and complete at the 160- and 250-F. levels. In storage, penicillin lost its potency faster in milk than in water.—*J. Dai. Sci., July, 1956.*

Simultaneous Crystal Violet Vaccine and Serum

To test the advisability of vaccinating swine simultaneously with crystal violet hog cholera vaccine and anti-hog cholera serum, in England, 20 pigs weighing about 60 lb. were injected subcutaneously in the ear with 1 cc. of vaccine, and intramuscularly with 30 cc. of serum. When these pigs were challenged with an injection of virus, a different pig on each of postvaccination days 5, 12, 14, 17, 21, 24, and 60, they remained normal; but the pigs injected on days 19 and 83 died of cholera while the 1 on day 106 reacted but recovered. The pigs challenged by contact infection on postvaccination days 5, 12, 17, 19, 21, and 60 remained normal, while pigs thus challenged on days 14, 24, 83, and 106 died. This method of vaccination was not recommended.—*T. M. Doyle in Vet. Rec., July 7, 1956.*

Crystal Violet Vaccine Immunity

In an epizootic of hog cholera in Japan, many animals vaccinated with crystal violet vaccine a year before resisted infection.

When nursing pigs, 20 to 21 days old, were injected subcutaneously with 3 ml. of crystal violet vaccine and the immunity of 3 was challenged a month later, 1 showed a moderate reaction, 2 no reaction; of 3 challenged after three months, 1 recovered after a moderate reaction and 2 died of cholera. Of 6 pigs vaccinated when 55 days old, 15 days after weaning, with 3 ml. of crystal violet vaccine, 2 withstood immunity challenge at 93 days, 2 at 183 days, but 1 of the 2 challenged at 273 days died of typical cholera. All control pigs died after challenge.—*Nibs Bull. Biol. Res. (Japan), Spring, 1956.*

Longevity of Cholera Virus in Pork

Pork from swine experimentally infected with hog cholera is always dangerous when raw, even after it has undergone some fermentation (decomposition). The virus is not inactivated by the smoke-curing process and it may live in sausage cooked at 75 to 82 C. for five minutes. It is destroyed at 80 to 85 C. for some smaller sausage in ten minutes, whereas other small sausages and all large sausages may require up to 45 minutes.—*Rev. de Path. Gén. et Physiol. Clin., May, 1956.*

Malignant Granulosa Cell Tumor in a Cat

EDWARD BAKER, V.M.D.

Englewood, New Jersey

Special tumors of the ovary, which include granulosa cell, theca cell, Brenner tumors, dysgerminoma, and others, have been observed from time to time in the dog but apparently have not been previously reported in the cat.[1]

According to Bloom,[1] the granulosa cell tumor is not uncommon in dogs and usually occurs after 5 years of age. This tumor usually affects only one ovary and ranges in size from several millimeters to 10 cm. in diameter. It is round or oval and may be lobulated on the outer surface; the cut surface may be solid but often shows cystic cavities filled with a clear or sanguineous fluid.

Microscopically, great variations occur even in the same tumor. The cells resemble the granulosa cell of the normal ovary and form microfollicular or macrofollicular follicles, whorls, trabeculae, cylinders, gyriform patterns, or sarcomatoid structures; the cells may be diffusely arranged as solid sheets or masses. Rosette formations resembling the Call-Exner bodies are not uncommon. At times, the granulosa cells may become luteinized and will then become larger, irregular, vacuolated, and polyhedral.

The tumor is usually benign or of low grade malignancy, but metastases may spread to the peritoneum and retroperitoneal lymph nodes. This description compares with that seen in women.[2]

Case Report

A 3-year-old domestic female cat was first examined on March 16, 1955. She apparently had normal estrous cycles, except that she seemed to be disinterested in the neighborhood toms. Following the last estrus, which occurred about two months prior to examination, a gradual swelling of the abdomen was interpreted as being due to pregnancy. Three days prior to examination, the cat developed anorexia and began to vomit. On examination, she was

Dr. Baker is a small animal practitioner in Englewood, N. J.

The author expresses his appreciation to the Armed Forces Institute of Pathology for its help in preparing a microscopic diagnosis in this case.

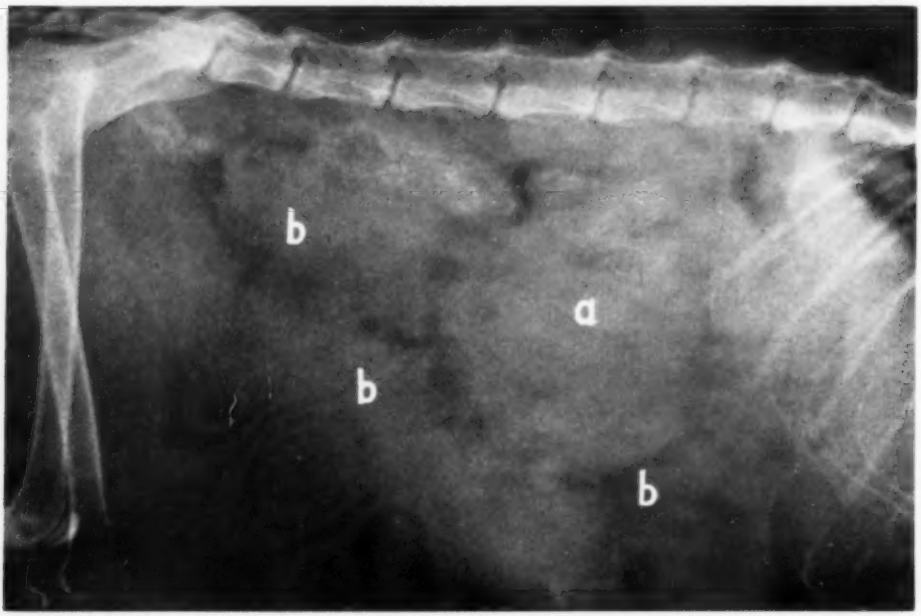

Fig. 1—Radiograph showing involvement in the abdomen of the cat: (a) large mass; (b) small masses.

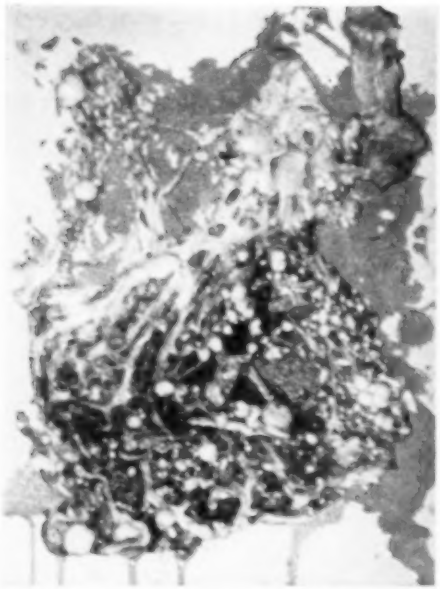

Fig. 2—Photograph of the omentum of the cat, showing tumorous nodules.

depressed and dehydrated, and a hard mass was palpated in the abdomen. The temperature was 101.4 F. A lateral radiograph clearly outlined a large central mass and several smaller masses in the abdomen (fig. 1). The owner requested euthanasia.

Gross Pathology.—When the abdomen was opened, large masses of gray-white nodules varying greatly in size and having a miliary distribution were seen. The omentum (fig. 2) was almost unrecognizable because of these nodules. Mushroom-like nodules of varying sizes were seen attached to the diaphragmatic peritoneum and, to a lesser extent, in the rest of the peritoneum. Retroperitoneally, on the dorsal wall of the abdominal cavity, and lying between the posterior pole of the right kidney and the pelvis was a large mass measuring about 6.0 by 2.5 by 2.5 cm. (fig. 3). The omentum was adherent to the small intestine at the pancreas.

The right ovary measured 8 cm. in diameter and had a lobulated appearance. This mass was mottled and ranged in color from white to blue-gray and red. In some

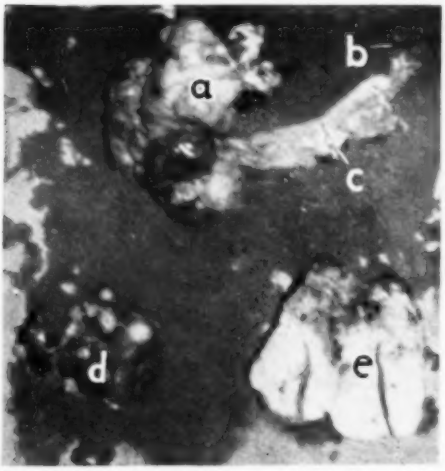

Fig. 3—Photograph of cat's organs showing neoplastic tissues: (a) tumorous left ovary connected to the normal right ovary (b) and showing excised body of uterus (c); (d) lung with tumorous nodules; and (e) neoplastic abdominal lymph node.

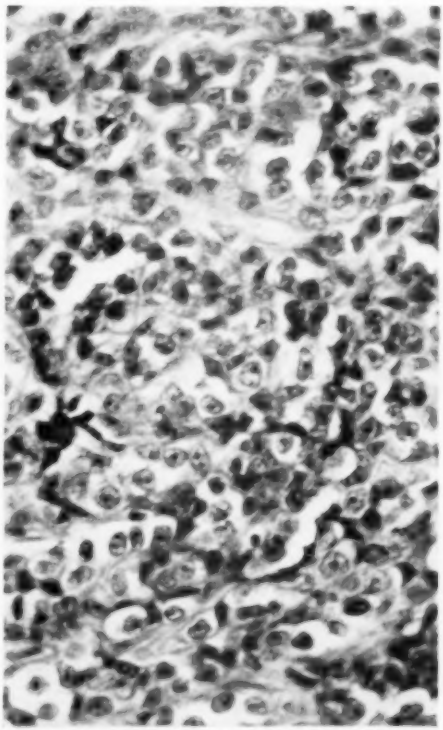

Fig. 4—Photomicrograph showing the characteristic cells of the tumor which were oval and round with a basophilic nucleus of medium size. The cytoplasm was scant, vacuolated, and stained poorly. x 435.

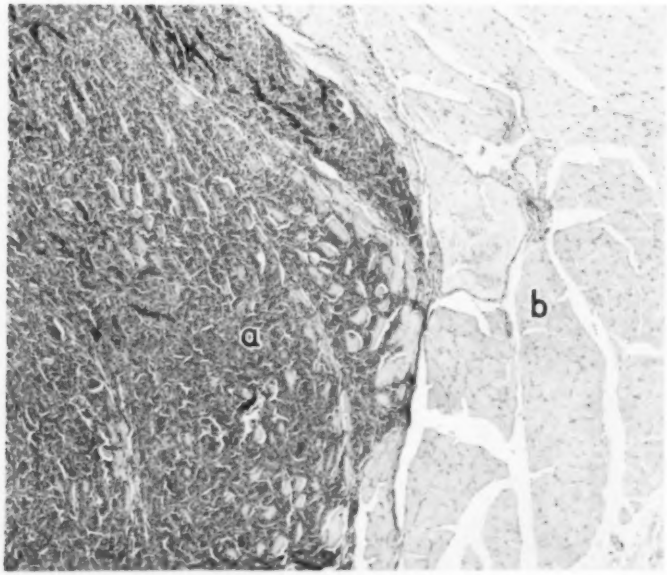

Fig. 5—Photomicrograph showing tumor (a) invading the abdominal muscles (b). x 48.

areas, the mass was firm and in others it had a cystic appearance. On cut surface, the same range of coloration was seen to extend into the mass. The cysts contained a sanguineous fluid. The left ovary was atrophic, and the left and right uterine horns appeared grossly normal.

The lungs contained small, grayish red nodules about 5 mm. in diameter. No other gross lesions were seen.

Microscopic Pathology.—No normal ovarian tissue was recognized. The characteristic neoplastic cell had an oval-to-round basophilic nucleus of medium size with a heavy chromatin net and a single nucleolus. The cytoplasm was scant, vacuolated, and stained poorly. The neoplastic cells at times formed and lined irregular glandlike spaces. The lining varied in thickness from one cell to many, the latter blending into solid masses of similar cells. The neoplastic cells frequently grew diffusely, being divided into small islets and irregular rows of one to several cells' thickness by an abundant fibrous stroma. The cell type and pattern of growth were suggestive of granulosa cells (fig. 4).

Digestive Tract.—No change of significance was recognized in the microscopic structure of the digestive organs.

Abdominal Wall.—The peritoneum was apparently initially involved and the skeletal musculature had been involved secondarily by extension and invasion by the tumor (fig. 5). The neoplastic tissue was essentially identical to that described in the right ovary.

Other Organs.—Metastatic neoplastic nodules, of the same type as previously described, were found in the lungs.

No neoplastic tissue was found in the liver, kidneys, spleen, and heart.

Diagnosis.—The diagnosis was granulosa cell carcinoma of the right ovary of a cat, with multiple metastases to the peritoneum and lungs.

SUMMARY

The multiple metastases in the peritoneal cavity were apparently the result of multiple seedings by way of peritoneal fluid, while the involvement of skeletal musculature, and perhaps regional lymph nodes, probably was due to secondary extension. The metastatic focus in the lung represented vascular spread.

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Chinchillas are not bothered by fleas because of their dense fur.— *Nat. Chinchilla Breeder, Aug., 1956.*

A Survey of *Thelazia Californiensis*, a Mammalian Eye Worm, with New Locality Records

WARREN E. PARMELEE, B.A.; ROBERT D. LEE, M.S.; EDWARD D. WAGNER, Ph.D.; HOWARD S. BURNETT, B.A.

Loma Linda, California

THE NEMATODE, *Thelazia californiensis*, is found most frequently parasitizing the exterior regions of the eyes of dogs and deer and less commonly the eyes of cats, sheep, coyotes, bears and human beings. Symptoms of such infections are excessive lacrimation and conjunctivitis. Infected dogs may be observed rubbing their eyes with their forepaws. Upon examination, whitish threadlike worms, 1 to 1½ inches long, may be seen gliding across the exterior surface of the cornea in characteristic serpentine movements. Large masses of these worms may be found in the conjunctival sac. In prolonged, heavy infections corneal opacities may be observed. These clear up following removal of the parasites, provided the scarification is not too extensive.

Before studying the biology and transmission, a survey of veterinarians was made for the purposes of studying the distribution of the parasite, alerting veterinarians to the problem, and establishing contact with the veterinarians so that living specimens could be obtained for transmission studies. The purpose of this paper is to review previously published reports of *T. californiensis* in California and to present new locality records for this state and two neighboring states.

Thelaziasis was first reported in this country by Allerton[1] in a dog near Los Angeles, Calif. Ackert identified the parasites as *Thelazia callipaeda*, a species common to the Orient. Price[2] recognized specimens from subsequent infections as a distinct species and proposed the name *Thelazia californiensis*. Since then, a number of workers have reported collections from other animals in several counties of California (table 1).

TABLE 1—Distribution of Thelazia Californiensis in California Based on Previously Published Reports and on the Results of this Survey

County	Published host records	No. of veterinarians*	Hosts reported in this survey
Alameda	Dog[3]	2	Dog
Butte	Dog[4]	1	Dog
Contra Costa†		1	Dog
El Dorado†		1	Dog
Fresno†		4	Dog
Glen†		1	Dog
Humboldt	Dog[4]	—	—
Kern†		1	Dog
Kings†		1	Dog
Lassen	Dog[4]	—	—
Los Angeles	Dog[1,2,4,5]	38	Dog, cat
Madera†		2	Dog
Marin†		2	Dog
Mariposa†		1	Dog
Mendocino	Dog[4]	1	Dog
Modoc†		1	Dog
Monterey	Dog[4]	3	Dog
Napa	Dog[2,4], cat[6]	1	Dog, cat
Nevada	Dog[4]	1	Dog
Orange	Dog[4]	—	—
Plumas†		1	Dog
Riverside	Dog[4]	4	Dog, horse
San Bernardino	Coyote[7]	5	Dog
San Diego	Dog[4]	11	Dog, cat, horse
San Francisco	Dog[4] man[8]	4	Dog
San Juoquin†		1	Dog
San Luis Obispo	Coyote[7]	1	Dog
Santa Barbara	Coyote[7]	4	Dog, cat
Santa Cruz†		3	Dog
Shasta	Dog[1,3]	1	Dog
Sierra†		1	Dog
Siskiyou	Dog[4]	—	—
Solano	Dog[4]	1	Dog
Sonoma	Sheep[9] deer[10]	5	Dog
Stanislaus†		2	Dog, horse
Sutter†		1	Horse
Tehama	Dog[4]	—	—
Tulare	man,[11] deer[12,13]	4	Dog, cat
Ventura†		2	Dog, cat

*Number of veterinarians reporting positively in this survey

†Counties not previously reported with T. californiensis.

During August, 1955, letters were sent to 1,230 California veterinarians requesting information on any cases of eye worms they had treated. Of 395 reporting, 42 were invalid because the replies were neither positive nor negative. Therefore, this survey is based on the returns of 353 veterinarians, 31 per cent of whom submitted

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This survey is part of work being done on a U. S. Public Health Service grant (E-951).

The authors thank the many veterinarians and others who responded to requests for information and specimens.

If the reader has additional data regarding the distribution of T. Californiensis, please contact the writers. To complete the biological studies now under way on this parasite, fresh and preserved specimens are needed. Fresh material is preferred and will be appreciated. Live specimens keep well when shipped in normal saline by air mail.

Fig. 1—The distribution of Thelazia californiensis in California based on the 1955 survey of veterinarians.

positive reports. Replies stating the locality in which the animal became infected are compiled according to county (table 1) and the geographical distribution is graphically portrayed (fig. 1).

Breeds of dogs examined were indicated by veterinarians in 81 instances (table 2). Also reported in the survey were 12 cats, 7 horses, and 1 human being.

The reason for the higher infection rates among the larger dogs may be due to either the distribution of the larger dogs in mountainous areas or to the individual temperament of the dog which may play a part in the tolerance of possible arthropod vectors in the eye region.

In cooperation with the California Department of Fish and Game, 129 deer were checked by the authors during November, 1955, at three widely separated points, all on Highway 395, in Mono County. The three check stations were at Coleville (35 mi. north of Bridgeport), Fales Hot Springs (17 mi. north of Bridgeport), and Tom's Place (26 mi. north of Bishop). Seven deer were found to be infected. This established a new county record.

That *T. californiensis* is not necessarily limited to California is borne out by a report supplied by Bischoff[14] of the California Department of Fish and Game Laboratory. Eleven eye worms were removed from the eyes of a deer by Bischoff and Brunetti* on Nov. 13, 1951, 2 mi. southwest of McCluskey's Ranch (Red Rock Canyon Rd.), Washoe County, Nevada. This is the first report of this parasite from Nevada.

Schneider[15] notified us of a case of eye worms in a dog from Galice, Ore. *Thelazia californiensis* has not been previously reported from Oregon, but there is no reason that it should not be found on both sides of the state line. Undoubtedly other cases have been seen, but not reported, in Nevada and Oregon. The distribution of this parasite may yet be shown to extend throughout several of the western states.

TABLE 2—Breeds of Dogs Reported by Veterinarians in this Survey; in Other Cases Breeds Were not Specified

Breed	No. reported	Breed	No. reported
Collie	20	Pointer	2
German Shepherd	18	Golden Retriever	2
Mixed	8	Long-Haired Toy	1
Terrier	8	Pomeranian	1
Hound	8	Whippet	1
Boxer	5	Chow Chow	1
Cocker Spaniel	5	Pekingese	1

SUMMARY

Thelaziasis is being recognized more frequently in wild and domestic animals. As a result of a recent survey made among veterinarians, 18 additional California counties have been added to the list of 22 previously reporting *Thelazia californiensis*. Only 18 of the 58 counties have not reported the infection. The neighboring states of Nevada and Oregon are now known to contain this parasite. One new case in a human being has been observed.

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*Federal Aid in Wildlife Restoration Project W-35 R.

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[13]Dixon, J. S., and Herman, C. M.: Studies on the Condition of California Mule Deer at Sequoia National Park. California Fish and Game Dept., *31,* (1945): 3-11.

[14]Bischoff, A. I.: Personal communication, 1956.

[15]Schneider, A. C.: Personal communication, 1955.

Piperazine for Ascarids in Chickens.—Piperazine compounds eliminated adult Ascaridia in chickens, in single doses of 100 to 500 mg. per kilogram of body weight, the dosage depending on the compound used. The drug, given in food or water, produced narcosis in the parasites which were carried to the exterior by normal peristaltic actions.—*Poult. Sci., May, 1956.*

Barn Roofs that Reflect Heat.—A roof covered with asphalt shingles or dirty galvanized steel will reflect approximately 11 per cent of heat from the sun's rays; one of new galvanized steel will reflect 34 per cent; but an aluminum roof painted with white lead will reflect 74 per cent. This is important in a henhouse since egg production is best at about 55 F., while heat prostration may occur when the temperature is above 95 F.—*Progressive Farmer, August, 1956.*

Four Deaths After BCG Vaccination.—Four deaths from tuberculosis in young persons, following BCG vaccination, were investigated. Histologically, the lesions were not typical of tuberculosis. The authors believe that "*Mycobacterium tuberculosis* had only the significance that *Hemophilus influenzae* has . . . in the course of influenzal pneumonia or that *Salmonella suipestifer* has in the course of hog cholera." Contamination with the organism may have occurred prior to vaccination.—*J.Am.M.A., July 28, 1956.*

Pulmonary Brucellosis in Man

In a survey on pulmonary brucellosis, only 13 physicians reported cases (11 acute and 30 chronic). Of the 41 cases, 38 (farmers, ranchers, and packing house workers) had contact with cattle, sheep, or swine. The infections were: *Brucella abortus* (83.7%), *Brucella melitensis* (11.6%), and *Brucella suis* (4.7%).

In chronic brucellosis, especially the *abortus* type, significant agglutination reactions are the exception rather than the rule. Blood culture may be negative, especially if the infection is well localized. The intradermal brucellergin test should be deferred until other tests are taken since it may stimulate a reaction.—*J.Am.M.A., July 21, 1956.*

Tuberculin Testing Young Americans

The rapid decline in human tuberculosis in the last decade is increasing the interest in the tuberculin test as a supplement for mass x-ray surveys. A tuberculin product (PPD), adopted by the National Tuberculosis Association and the World Health Organization, is used.

In 1949 to 1951, more than 120,000 white Navy recruits and college students were tested, using 0.1 ml. (0.0001 mg.) of PPD intradermally. In 48 to 72 hours, the diameter of the reaction lesion was measured, 5 mm. or more being considered positive. The average frequency of reactors was 8.8 per cent, corresponding to an annual infection rate of less than 5 per 1,000 during the last 20 years. The infection rate was highest (20%) in the southwestern states and lowest (4%) in the northern states; it also was higher in city than in farm residents. Many light reactions were ascribed to a low-grade sensitivity not related to tuberculosis infection.—*Pub. Health Rep., July, 1956.*

Lung Cancer in Dogs.—The increase in lung cancer in man is little greater than in dogs (in Holland). Necropsies on 9,781 dogs in 30 years, to 1954, revealed lung cancer in 22, 16 of them in the last four years. This could not be accounted for by the greater length of life of the dogs, nor by improved diagnosis since the tumors were usually large. Most were of the cylindrical carcinoma, not the epidermoid, type of lung carcinoma usually encountered in man.—*J.Am.M.A., July 21, 1956.*

Equine Euthanasia

NORMAN C. ROBERTS, D.V.M.

San Diego, California

For several centuries, veterinarians have been attempting to destroy horses humanely. Practitioners engaged in Thoroughbred, Standardbred, and show horse practice are frequently confronted with the need to destroy animals injured during a race or while working. Often, this must be done in a conspicuous place, in the presence of a curious and unenlightened group of onlookers, so it is of paramount importance to avoid any struggle or pain.

Many methods of euthanasia are available, but most of them are unsatisfactory when performed before the general public. Through the years, destruction with a pistol has probably been the most satisfactory method. Shooting an animal in the brain is efficient and rapid provided a large-caliber pistol is used; however, it is dangerous when undertaken by an inexperienced person or in a congested area. It is extremely embarrassing to miss the target and have the bullet lodge in the sinuses, resulting only in a profuse hemorrhage from an animal which must be shot again under a less ideal situation. In addition, there has been objection to this means of euthanasia from the public.

Strychnine solution has been used intravenously, but it is difficult to get a highly lethal quantity of the drug into a small amount of solution. The animal is completely conscious during the violent convulsions which may last several minutes.

Chloral hydrate or magnesium sulfate and combinations of the two are bulky and slowly effective when used intravenously, and there is invariably considerable struggling before a fatal dose is injected.

Intravenous solutions of barbiturates have proved to be the most satisfactory for this purpose in our race track practice. Recently, I have been using a solution containing two toxic barbiturates, sodium N-amylethylbarbiturate and sodium sec.-butylethylbarbiturate,* and have found it most efficient and satisfactory for equine euthanasia.

Dr. Roberts is an equine practitioner in San Diego, Calif.

*Lethol, Pitman-Moore Co., Indianapolis, Ind.

ADMINISTRATION

A 100-cc. glass syringe with a Luer-Lok attachment is preferred. Two 50-cc. syringes will prove satisfactory but both should be filled prior to the operation. In the event of cold weather, the product should be placed under the hot-water tap until thoroughly warm in order to render it less viscid. A 14-gauge needle is introduced into the jugular vein, and 100 cc. of solution is injected as rapidly as possible. If two syringes are used, it is important to make the change with a minimum of delay. The injection should not require over 30 seconds, and the horse must be given the entire dose before going down. The needle is withdrawn at the completion of the injection.

The animal will invariably collapse within one minute after the injection is started. The collapse is dramatic and without evidence of pain, since the horse is anesthetized by the time it falls to the ground. The animal seldom struggles and never attempts to rise or strike his head on the ground. Death, as judged by cardiac failure, occurs within a few minutes.

REPORT OF CASES

During the past year, I have destroyed 23 horses by this method. Most of the animals had some type of fracture, usually compound. Their ages varied from a yearling to a 16-year-old gelding. Only 2 of these animals struggled following the injection; the movements were involuntary and neither showed signs of consciousness or tried to rise. One of the 2 died approximately ten minutes following collapse, no additional treatment being necessary; the other was given an additional 50 cc. of solution before death ensued.

With this solution, a veterinarian can perform euthanasia in a highly professional manner. The type of death is certainly not unpleasant to anyone who chances to observe the procedure. The 100-cc. dose is adequate for most horses.

SUMMARY

Twenty-three horses, varying from a yearling to a 16-year-old gelding, were destroyed humanely by the intravenous injection of 100 cc. of concentrated barbiturate solution through a 14-gauge needle placed in the jugular vein. The injection is given in 30 seconds. The animals collapse within one minute after completion of the injection.

What Is Your Diagnosis?

Because of the interest in veterinary radiology, a case history and accompanying radiographs depicting a diagnostic problem are usually published in each issue of the JOURNAL.

Make your diagnosis from the picture below—then turn the page ▸

History.—A female, Collie-type dog, 10 years old, with paralysis of the hindlegs, urinary bladder, and anal sphincter was submitted for examination. A radiopaque dye was injected into the subarachnoid space at the cisterna magna and a myelogram of the posterior thoracic and anterior lumbar regions was taken.

Figure 1

(Diagnosis and findings are reported on next page)

Here Is the Diagnosis

(Continued from preceding page)

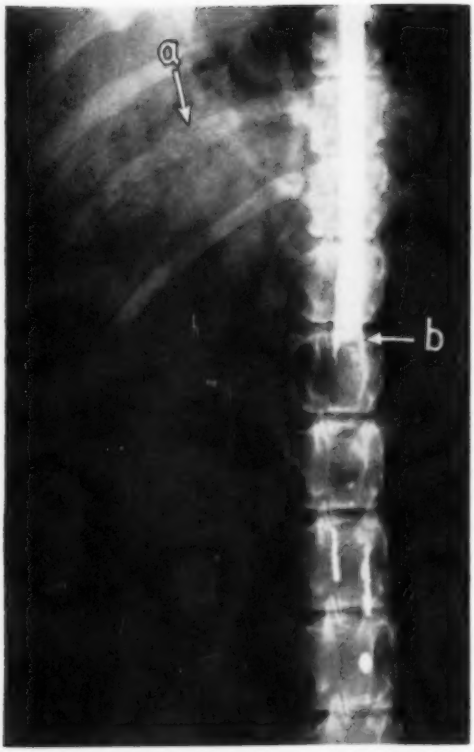

Fig. 2—Radiograph showing (a) ossification of the tissue in the thoracic area and destruction of the involved ribs, and (b) a subarachnoid block of the spinal fluid at the second lumbar vetebra.

Diagnosis.—A blockage of the subarachnoid space near the junction of the first and second lumbar vertebrae as indicated by the arrested flow of the injected opaque material. Also ossification of the soft tissues of the thorax and degeneration of the tenth, eleventh, and twelfth ribs (shown on left) in the posterior thoracic region just lateral to the spinal column.

Comments.—At necropsy, a pachymeningitis was found where the spinal blockage occurred at the second lumbar vertebra, and a tumor was found invading the involved thoracic area. The primary tumor probably originated in an adrenal gland or an ovary.

Our readers are invited to submit case histories, radiographs, and diagnoses of interesting cases which are suitable for publication.

This case was submitted by Drs. William I. Gay and Ladd Loomis, National Institutes of Health, Bethesda, Md.

Correction in "What Is Your Diagnosis?"—Our attention has been directed to errors in the editor's comments in "What Is Your Diagnosis?" (Aug. 15, 1956: 148). Obviously, since the exostosis was confined to the point of insertion of the extensor tendon, it should not be called a "ringbone"; furthermore, it was not associated with "collateral ligaments" as unfortunately intimated in a poorly chosen quotation.—ED.

The Incidence of Gastrointestinal Nematodes in Illinois Cattle

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ALTHOUGH gastrointestinal nematodes are known to be of major importance in midwestern sheep, their role in midwestern cattle is not well known. In order to obtain information on this subject, a survey was undertaken during the summer of 1955. Fecal samples were collected from 135 cattle on 24 randomly selected farms in the vicinity of Urbana in east central Illinois. Most of the fecal samples were from adult animals, but some yearlings and a few calves were included.

METHODS

Egg counts were made by the McMaster technique.[2] A dilution factor of 200 was used, *i.e.*, each egg counted represented 200 eggs per gram of feces (e.p.g.). Since a maximum of four counts was made on each sample, the lowest egg count recorded (represented by a single egg in one of the counts) was 50. If the animals had fewer eggs than this, their counts were recorded as zero. The average egg counts recorded for the 5 or 6 animals examined from each farm are, therefore, possibly somewhat lower than the actual number present, but this does not affect their usefulness in determining the incidence of significant parasitism, since such low counts indicate light infections.

RESULTS

The results recorded (table 1) are only for strongylinate eggs (*i.e.*, eggs of members of the suborder Strongylina which includes the two most important groups of ruminant gastrointestinal parasites, the superfamilies Strongyloidea and Trichostrongyloidea), exclusive of Nematodirus. Except for the latter, these eggs all look much alike.

In the present survey the only other eggs encountered were those of Nematodirus spp. and Trichuris spp. and both were rare. Nematodirus was found in 2 of the animals (1.5%), and Trichuris in 2 others (1.5%). The fecal egg count in each of the 4 animals was 50, and the mean egg count for each species in all the cattle was 0.7 e.p.g.

The mean egg count of the 65 adult dairy cows was 58 e.p.g., and the range was 0 to 700. In the 45 adult beef cattle, the mean was 43 e.p.g. and the range 0 to 400. The counts were higher in the yearlings, averaging 162 e.p.g. for the 8 dairy animals and 604 e.p.g. for the 13 beef animals. This last figure is misleading, however, since it is due to a single herd (J) in which exceptionally high counts were encountered.

Too few calves were examined for significance, but the mean of the four egg counts which were made was 300.

DISCUSSION

It is well recognized that because of their variability and because of a number of

TABLE 1—Numbers of Strongylinate* Eggs per Gram of Feces in Illinois Cattle, Summer, 1955

Type of cattle and age	Farm No.	No. of animals	No. strongylinate eggs/gram feces: Range	Mean
Dairy, adult	A	6	0 to 150	33
	B	6	0	0
	C	5	0 to 200	50
	E	6	0	0
	G	5	0 to 400	140
	H	5	0 to 100	20
	I	6	0	0
	K	6	0 to 700	116
	N	6	0 to 150	25
	O	6	0 to 400	150
	P	6	0 to 100	33
	R	5	0 to 50	30
	T	6	0	0
	U	2	150 to 250	200
Total or mean		65	0 to 700	58
Dairy, yearling	C	1	350	350
	F	6	0 to 250	92
	R	1	400	400
Total or mean		8	0 to 400	162
Beef, adult	H	5	0 to 100	20
	L	6	0 to 50	8
	M	5	0 to 100	20
	Q	6	0 to 50	17
	V	5	0 to 400	150
	W	6	0 to 200	33
	X	6	0 to 350	92
	Y	6	0 to 50	17
Total or mean		45	0 to 400	43
Beef, yearling	O	6	0 to 100	33
	J	6	300 to 2,800	1,275
	U	1	0	0
Total or mean		13	0 to 2,800	604
Beef, calves	G	1	100	100
	H	1	200	200
	M	1	400	400
	V	1	500	500
Total or mean		4	100 to 500	300

*Exclusive of Nematodirus.

From the College of Veterinary Medicine and Agricultural Experiment Station, University of Illinois, Urbana.

factors which can affect them, single fecal egg counts give only a rough idea of the extent of infection in an animal or in the herd. Egg counts are nevertheless of great value, particularly if, as in the present case, several animals are examined in each herd.

The interpretation of egg counts has been discussed by a number of workers. Roberts *et al.*[1] dealt in some detail with the problem in cattle. While in some cases, animals with extremely low counts may be suffering from clinical parasitism, the following numbers of eggs/gram of feces may be taken to indicate borderline pathogenicity for different nematode species: *Bunostomum phlebotomum*, 300; *Trichostrongylus axei*, 400; *Haemonchus placei* (*H. contortus* of cattle) and *Oesophagostomum radiatum*, 500; Cooperia spp., 10,000. It would, therefore, appear highly conservative to take 300 mixed strongylinate eggs/gram of feces as a level suggestive of borderline or subclinical infection.

None of the adult dairy or beef herds had average egg counts as high as 300; the highest was 200. Only 4 of the 65 dairy and 2 of the 45 beef adults had counts of 300 or more; the highest was 700.

Eight of the 21 yearling dairy and beef animals had counts of 300 or more. Six of these were in a single beef herd (J) in which the counts ranged from 300 to 2,800, with a mean of 1,275. Peculiarly enough, these animals appeared in top condition, suggesting that their high counts may have been due to a relatively nonpathogenic species such as Cooperia.

The average egg count in the 4 calves studied was 300; two of them were above this figure.

It may be concluded from this study that adult dairy and beef cattle in east central Illinois do not in general suffer from serious parasitism, although a few individual animals (5.5% in the present survey) may have 300 or more strongylinate e.p.g., the level taken as suggestive of borderline or subclinical infections. In addition, an occasional herd may be heavily infected. Adult animals may, however, serve as a source of infection for young ones grazing with them.

The average egg count in yearling animals was above 300 in one of the three herds from which more than 1 animal was examined. More yearlings and calves should be examined before solid conclusions can be drawn on the extent and seriousness of their parasitism.

SUMMARY

In a fecal survey of 135 cattle on 24 farms in east central Illinois, averages of 58 strongylinate eggs per gram of feces (e.p.g.) were found in 65 adult dairy cattle, 43 e.p.g. in 45 adult beef cattle, 162 e.p.g. in 8 dairy yearlings, 604 e.p.g. in 13 beef yearlings, and 300 e.p.g. in 4 beef calves. Nematodirus and Trichuris eggs were found in 2 animals each. Egg counts of 300 e.p.g. (the level taken as suggestive of borderline or subclinical infection) were found in 6 of the adult animals, 8 of the yearlings (of which 6 were in 1 herd), and 2 of the calves. It is concluded that adult cattle in this area do not in general suffer from serious parasitism.

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Ammonium Chloride as an Agent to Acidify Bovine Urine

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Animals which consume only plant food have a surplus of fixed base : sodium, potassium, and calcium. Freed from organic salts by oxidation, the excess of this base is excreted in the urine, largely in the form of carbonates and bicarbonates. It is these salts, dietary in origin, that are responsible for the characteristic alkaline reaction of the urine of herbivores.

Therapeutically, it is sometimes desirable to acidify the urine. Some infections of the urinary tract are controlled simply by rendering the urine acid; some urinary antiseptics, notably methenamine and mandelic acid, are effective only in an acid

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This study was supported in part by a student research scholarship from Lederle Laboratories Division, American Cyanamid Co., Pearl River, N. Y.

Journal series paper No. 1618, approved by the director of the Missouri Agricultural Experiment Station, Columbia.

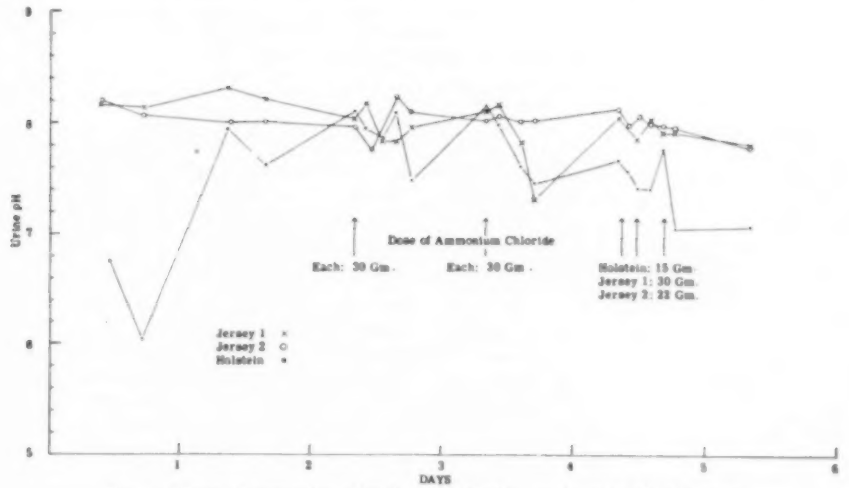

Fig. 1—Effects of ammonium chloride administration on the urine pH of 3 cows.

urine. Ammonium chloride is frequently recommended[1-3] as an acidifying agent; however, there have been no careful studies to determine the amount of this agent necessary to render the urine consistently acid.[3]

MATERIALS

Three mature animals were studied; 2 were normal Jersey cows, weighing approximately 800 lb. each, and 1 was a cow with a rumen fistula, a Holstein-Friesian weighing approximately 850 lb. The cows had access to water and alfalfa hay at all times and were fed 3 lb. of a grain mixture consisting of equal parts of wheat bran, oats, and ground corn, twice a day. Ammonium chloride was given in gelatin capsules. Urine was obtained at various intervals (fig. 1 and 2) by stroking the vulva; pH was measured with a Beckman, model G, pH meter.

RESULTS AND DISCUSSION

The administration of comparatively large amounts of ammonium chloride had no apparent ill-effect on the 3 cows. There were no signs of acute pulmonary edema, a condition produced in the guinea pig, rat, and cat by ammonium salts[4]; nor was there evidence of the gastric irritation reported in carnivores.[2] The appetite of the cows was not affected; each consumed an average of 13 lb. of hay per day during a two-day control period and during the first three days of drug administration. Feed

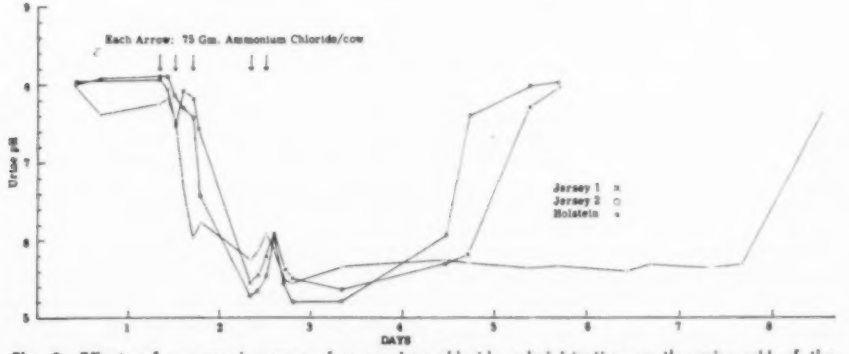

Fig. 2—Effects of a second course of ammonium chloride administration on the urine pH of the 3 cows.

consumption remained normal during the entire experiment.

The administration of one dose of 30 Gm. of ammonium chloride on two successive days, followed on the third day by the administration of 45, 66, and 90 Gm. in three divided doses (fig. 1), did not acidify the urine of any of the cows. This may have been because the capsules were passed into the rumen (confirmed in the cow with the fistula) and only a dilute solution of ammonium chloride was absorbed over a long period of time, or it may have been because the nature of the cow's diet makes her more resistant to acidifying agents than are other species.

After a four-day rest, each cow was given three 75-Gm. doses of ammonium chloride the first day and two 75-Gm. doses of ammonium chloride the second day. Four hours after the first dose, there was a decline in the urine pH and, within 12 hours, the urine was acid in all 3 cows (fig. 2). The speed of these changes is somewhat surprising and indicates either absorption from the rumen or else a rapid passage of water from the rumen to the lower parts of the digestive tract.

The two urine samples taken at six-hour intervals from the cow with the rumen fistula during the first control period were acid. At this time, some difficulty was experienced in keeping the fistula plugged. During both experimental periods, this cow seemed more sensitive to ammonium chloride than the normal cows; this may have been related to the degree of fill, since she lost rumen contents at irregular intervals. Three days after the fifth dose of ammonium chloride, this cow developed frank hematuria lasting approximately 24 hours; otherwise, she was normal.

SUMMARY

Ammonium chloride in usual doses (30 to 90 Gm. daily) for three days did not acidify the urine of 3, 800-lb. cows. The administration of 225 Gm. of ammonium chloride in divided doses in one day did acidify the urine; changes in urine pH were noticed within four hours and persisted for 48 hours in 2 normal cows. No untoward symptoms were caused by the administration of comparatively large doses of ammonium chloride.

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Copper Sulfate and Moniliasis.—Copper sulfate was ineffective in moniliasis (crop mycosis) in chicks and poults when infection was produced by oral inoculations with *Candida albicans*. Two of five drug trials resulted in a more favorable environment in the crop for this infection.—*Poult. Sci., May, 1956.*

A Practitioner, on Atrophic Rhinitis

How atrophic rhinitis first appeared in a province in Quebec in 1947, and how it was spread in practical experiments, was related by L. A. Gendreau, D.M.V., Sherbrooke, Que. Apparently introduced by boars, the disease was transmitted by moving infected animals into clean herds. The disease then spread rapidly.

The symptoms, which first appear in pigs 2 to 3 weeks old, are: sneezing, sniffling, slight nasal hemorrhage, rubbing the nose, shaking the head, and often coughing and signs of pneumonia. At 8 to 10 weeks of age, distortion of the snout becomes apparent and deaths (up to 20%) start to occur. Half the herd may develop deviations of the snout but close observation may detect sneezing in nearly all of the animals. Growth is retarded in nearly all cases showing clinical symptoms. When growth of the upper jaw is arrested, resulting in no incisor and poor molar contact, the pig can not eat properly and about half may never reach market weight. Deformity of the hard palate and turbinate bones results in inhalation of dust and feed, which may produce lung abscesses and pneumonia.

The disease has been transmitted by apparently healthy pigs from an infected herd. Heredity and malnutrition seem not to be factors in susceptibility. Necropsies reveal: pneumonia, adhesions of the lungs to the costal and diaphragmatic pleura, abscesses in the lungs and throat region, adhesions of the pericardium to the heart and pleura, as well as the changes in the nasal cavity.—*Vet. Sci. News, Univ. of Wisconsin, Summer, 1956.*

Systemic Amebiasis with Distemper in a Dog

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NATURAL INFECTION of dogs with *Entamoeba histolytica* has been reported by several authors in different parts of the world.[1-4,6,7] The lesions described have been confined to the gastrointestinal tract, mainly in the large intestine. Infections with *E. histolytica* in man are also usually centered in the large intestine, but invasion of other organs is not uncommon. The most common forms of extra-intestinal amebiasis in people are hepatic, pulmonary, juxta-intestinal, cutaneous, and genital. Amebic abscesses of the brain have been found in 1 per cent of fatal cases in people, and numerous other tissue sites of metastatic amebic infection have also been described.

Extra-intestinal amebiasis has not been reported in dogs with natural infections, but one instance was seen in a dog with an experimental infection.[5] The dog, infected with cysts from a case of chronic amebiasis in man, developed an intestinal amebiasis of mild degree. It died suddenly and, on necropsy, was found to have multiple amebic abscesses of the liver, with perforation into the pleural cavity and involvement of the right lung.

CASE REPORT

The purpose of this article is to describe a case of systemic amebiasis in a dog that was also affected with distemper. Two hound litter mates, 3½ months old, were vaccinated on Feb. 26, 1956, with chicken embryo distemper vaccine and desiccated hepatitis vaccine in conjunction with anti-distemper hepatitis serum. Five days later, a third litter mate was likewise vaccinated. Ten days after the first 2 were treated, they had 1 to 2 degrees of fever, were coughing, and showed a purulent discharge from the eyes and nose. The sicker dog had a "mushy" stool and a light hookworm infection and developed labored breathing and a moist cough just before death 15 days following vaccination. The other dog had a marked diarrhea and a moderate hookworm infection. Dog 3 developed mild symptoms of distemper. The latter 2 dogs apparently recovered. At the latest report, 25 days after vaccination, both animals were doing better, but 1 was still receiving tetracycline. Tissue specimens from the dog that died were forwarded to this laboratory.*

Routine histopathological examination was made of sections of lung, liver, spleen, kidney, and urinary bladder. Numerous cytoplasmic inclusions characteristic of distemper were found in the epithelium of the urinary bladder. Since this observation furnished an apparent diagnosis, the initial examination was routinely completed. Other lesions noticed were some pneumonia, a few cytoplasmic inclusions in the epithelium of the bronchioles, and a few foci of lymphocytic and large mononuclear cell infiltration in the renal cortex, along with one glomerulus showing marked hyaline change and a thrombus in the afferent arteriole. The hyalinized glomerulus contained approximately 30 mononuclear structures, each with a small nucleus and abundant cytoplasm.

These structures were morphologically typical of *E. histolytica* as the organism appears in tissue sections. That observation prompted a careful restudy of the case.

The pneumonia was of a focal metastatic character, rather than bronchopneumonia from secondary bacterial infection characteristic of acute fatal cases of distemper. The lung tissue contained vaguely defined large foci of acute inflammation distributed without any particular relationship to bronchioles. In the pneumonic foci, the capillaries were congested and the alveoli were filled with a variable mixture of leukocytes and mononuclear cells of different types. Among the cells of the exudate, there were numerous structures morphologically typical of *E. histolytica* (fig. 1). The organisms ranged in size from 7 to 15 μ and each had a nucleus about 3 μ in diameter. The nucleus displayed a peripheral ring of chromatin and a centrally placed karyosome. Some of the parasites had small or medium sized cytoplasmic vacuoles, and many contained particles of iron-free, brownish pigment resembling acid formalin hematin precipitate seen in some of the tissue cells. Erythrocytes were also seen in some of the organisms.

In the center of several of the larger pneumonic foci, the alveolar walls and intra-alveolar cellular exudate were becoming necrotic. Between the pneumonic foci, there was some congestion and edema, but only a small amount of intra-alveolar cellular exudate. The large number of amebae in

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Approved by the Committee on Publications, School of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, publication No. 612.

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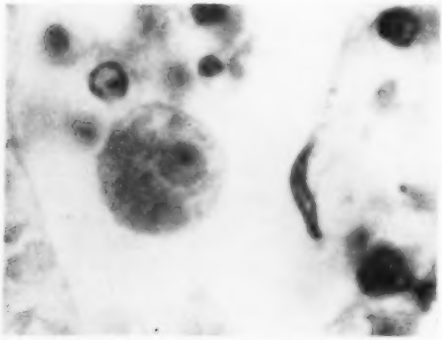

Fig. 1—Ameba and cellular exudate in pulmonary alveolus. x 1,800.

the pulmonary tissue and their obvious association with the pneumonic foci left no doubt that these protozoon parasites were a major factor in the cause of the pneumonia.

Evidence of amebic infection was found in the liver and spleen sections only by thorough examination under high magnification. There were scattered, solitary amebae in the liver, located either in the sinusoids or within hypertrophic Kupffer cells. No abscesses were seen, nor was there evidence of parenchymal damage or other lesions that could be attributed to the parasites. Scattered solitary amebae were also seen in the spleen, but in lesser numbers than in the liver and also without associated tissue reaction. The diagnosis on the basis of the above histopathological observations was systemic amebiasis with distemper.

DISCUSSION

This is apparently the first reported case of natural systemic infection with *E. histolytica* in the dog. Organisms morphologically characteristic of this ameba were found easily in the lungs and with some difficulty in the liver, spleen, and kidney. The pneumonia described was caused by the invasion of the lungs by the ameba. It was unfortunate that other tissues such as the large intestine and brain were not seen but since distemper was suspected as the cause of death, only liver, lung, spleen, kidney, and urinary bladder were sent for histopathological examination.

Natural intestinal infection in dogs with *E. histolytica* is not uncommon[1-4,6,7] but, except for an experimentally induced case,[5] no instance of systemic amebiasis has been recorded. Since intestinal infection in dogs has been reported to be as high as 8 per cent,[3] it seems unusual that more cases of extra-intestinal amebiasis have not been seen. It is difficult to produce, experimentally, an invasion of organs other than the intestine in dogs. It is, therefore, tempting to speculate that, in this case, distemper may have played a role in destroying resistance to invasion.

In the absence of sections of intestine, it is assumed that there was primary intestinal amebiasis in this animal.

It is possible that direct smears of the feces of this dog in 0.85 per cent saline might have demonstrated the amebae before death. Except for one report,[7] the cysts of *E. histolytica* have not been seen in feces from infected dogs. Since only trophozoites are commonly found in the dog, any procedure such as zinc sulfate, sugar, or salt flotation would destroy the organisms and consequently fail to provide evidence of infection.

Since cysts are probably not passed by dogs and since the trophozoites are fragile organisms, it is probable that infection of this and other dogs was not from other infected dogs but from the cysts in the feces of man. In one epizootic of natural infections in dogs,[3] a native "dog-boy" was found to be a carrier of *E. histolytica* and had typical cysts in his stool. Since this individual was the cook who prepared food for the pack, the outbreak of amebic dysentery in the dogs was attributed to him.

SUMMARY

A case of concurrent systemic amebiasis and distemper in a dog is described. Organisms characteristic of *Entamoeba histolytica* were found in large numbers in the lungs, with pneumonia. Amebae also were seen in the liver, kidneys, and spleen. Tissue specimens of the intestines were not available for examination.

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Psittacosis Diagnosis in Parakeets.—Diagnosing psittacosis in parakeets is most accurately done by isolating the virus from the tissues, but this requires killing the bird. In a recent test with 125 parakeets, reactions to the complement-fixation test, which had proved its value with human serums, was closely correlated to virus isolations. One millimeter of blood was obtained in a syringe (using a 25-gauge, ½-inch needle) from the jugular vein of the bird while it was anesthetized with ether.—*Pub. Health Rep., July, 1956.*

Sulfaquinoxaline Toxicity in Chickens.—Hemorrhagic disease in chickens, characterized by a prolonged bloodclotting time, by hemorrhages, and by necrotic lesions in the spleen, was produced by feeding sulfaquinoxaline. The resulting increased bloodclotting time was corrected when injections of vitamin K were given, but spleen lesions were not reduced. This indicates that sulfaquinoxaline has a toxicity in addition to its inhibition of intestinal synthesis of vitamin K.—*Nutr. Rev., Aug., 1956.*

Toxoplasma in Chickens.—Toxoplasma were found in 35 hens, in 26,000 necropsies in Denmark. In 11 of the 35, there was necrosis of the optic chiasma. The diagnosis was confirmed in six of the 21 affected flocks by serological or transmission tests.—*Vet. Bull., July, 1956.*

Toxoplasma gondii have been preserved for 209 days by slow freezing in a solution of 5 to 10 per cent glycerol. This avoids the cumbersome and dangerous method of frequent animal transfer and also preserves the organism unmodified by rapid animal passages.—*J. Parasitol., August, 1956.*

Trichinella in Alaskan Mammals.—In Alaska, where trichinosis is not uncommon, tissue samples from 2,433 mammals, representing 42 species, revealed *Trichinella spiralis* in 23 species and in 285 (11.7%) of all the animals. In various surveys since 1949, infection was found (21.7 to 52.9%) in grizzly bears, black bears, polar bears, dogs, wolves, foxes, ermine, wolverines and lynx, and in coyotes (12.5%) and 2 weasels (100%). Infection of 4.3 per cent or less was found in snowshoe hares, ground squirrels, red squirrels, voles, beavers, white whales, walrus, seals, and one species of flesh-eating birds.—*J. Parasitol., June, 1956.*

Diagnosis of Virus Pneumonia of Pigs

The principal histopathological finding in pigs intratracheally inoculated with the etiological agent of virus pneumonia of pigs (V.P.P.), as well as in animals naturally affected, was an extensive lymphoid hyperplasia of the peribronchial, peribronchiolar, and perivascular tissues. Lesions selected with the naked eye as typical of V.P.P. showed such a variation when examined microscopically that additional causes of the lesions seemed probable. The histopathology seemed to offer the more specific method of diagnosis.—*I. H. Pattison in Vet. Rec., July 28, 1956.*

Virus Pneumonia of Pigs.—Typical lesions of V.P.P. were recently found in 74 per cent of the hogs marketed at a packing plant in an eastern state; the infectious agent was isolated and the disease was reproduced. A similar disease and infectious agent has now been reported from Nebraska. It is estimated that 50 per cent of U. S. swine may be affected.—*Norden News, July, 1956.*

Virus Pneumonia in Canadian Swine

A lung infection closely resembling virus pig pneumonia has been demonstrated in swine in five provinces of Canada. The pathological changes suggest the activity of a virus rather than of bacteria or other agents, and bacteriological cultures were frequently negative or contained nonpathogenic organisms.

Since infection with viruses and strains of PPLO most likely occur during the first few days of life, much experimental work in the respiratory diseases of swine in the past is of doubtful value. In the future, only pigs born by cesarotomy or farrowed without contamination should be used in experimental work.—*F. W. Schofield in Canad. J. Comp. Med. and Vet. Sci., July, 1956.*

Rumen Sampling for Field Diagnosis

Samples of rumen fluid, for aid in diagnoses, may be readily obtained with a stomach tube. A stiff rubber tube, kept coiled so that its curve will facilitate passage, is passed through the mouth, often without a speculum. The tube for small calves is ½ inch in outside diameter and 3 ft. long; that for older animals may vary up to the 1½-inch Kingman tube. A smooth water hose 7½ ft. long is usually satisfactory.

When well into the rumen, if the curved tube is rotated, the end may be felt through the flank. For best results, the end should be below the usual site for rumenotomy. A back-and-forth motion encourages rumen contraction and flow of liquid. When the flow stops, it may be restarted by a pause, which allows liquids to collect around the end of the tube, or by an upward pressure on the lower left abdomen.

The disturbing conditions found may be: a gas bloat, frothy mixture, lack of liquid, or too much liquid. Milk or milk curd found in the rumen of a calf more than 4 days old usually indicates a pathological condition; it usually has been regurgitated because of an abomasitis. Blood clots have been recovered in acute traumatic gastritis. Some poisons can be detected by their odors.

The rumen microorganisms may be studied. Some individual, larger protozoa can be seen with the naked eye as small, light-colored particles resembling tiny bubbles but differing in that their motion in the fluid is erratic, not rising like a bubble. Normal protozoan content usually means a normal bacterial content also. However, the feeding of much grain may interfere with the bacteria and not with the protozoa.—*W. D. Pounden, D.V.M., Ohio, in Vet. Sci. News, Univ. of Wisconsin, Summer, 1956.*

Feeding hay or silage to cows on good pasture may provide some insurance against bloat but usually does not increase milk production or body weight. The cows will usually eat 5 lb. or more of hay per day but they will eat less grass.—*Hoard's Dairyman, Aug. 10, 1956.*

Hypervitaminosis A in Man.—Clinical hypervitaminosis A is often reported in children, but only four times previously in adults. A 21-year-old woman had taken 160,000 to 180,000 units of vitamin A daily for seven months. She had developed bone and joint pains, fatigue and insomnia, swelling of the ankles, ecchymosis, soreness of the mouth with blisters on the tongue, and frequent urination, but not loss of hair or weight, skin changes, headaches, or exophthalmos which are frequently found. Symptoms and signs disappeared promptly when the vitamin A was discontinued.—*J.Am.M.A., July 21, 1956.*

Feeding Enzymes to Young Pigs.—Adding pepsin, obtained from the stomachs of healthy hogs at packing plants, at the rate of 5 lb. per ton of feed makes possible the substitution of vegetable proteins such as soybean oil meal for part of the more expensive milk protein in starter feeds for pigs up to 5 weeks of age. Enzymes are secreted in sufficient quantities by older pigs.—*Wallaces' Farmer, July 21, 1956.*

Digestive Enzymes in Young Pigs.—The amylolytic activity of extracts of the pancreatic gland increases markedly with the advancing age of unweaned pigs. The lipolytic activity is high at birth and remains so. Lactase activity is high the first two weeks of life then declines rapidly; sucrase and maltase activity increase from negligible at birth to maximum at about 25 days. Thus, the suitability of different carbohydrates for young pigs must differ markedly.—*Vet. Bull., July, 1956.*

Feeds for Early Weaned Pigs

Pigs could not be weaned profitably, under Wisconsin farm conditions, before 3 weeks of age, and 4 weeks seemed better. The objections to earlier weaning were: poor appetites, scours, and increased difficulties and costs of feeding. When 3 weeks old, pigs made the transition from sows to dry rations satisfactorily but their growth was ordinary and the cost excessive.

Early weaning studies indicated that a simple dry ration of ordinary feeds was the most economical but that the pigs did not continue to grow as well as those that remained longer on the sows.—*Univ. of Wisconsin Release, Aug. 20, 1956.*

When fats are added to feeds for poultry and swine, they increase the energy content and caloric density, but this makes necessary a proportionate increase in essential amino acids, especially methionine. —*Proc. Am. Feed Mfr. A., May 23, 1956.*

Leunis Van Es, Veterinary Scientist

The death* of Dr. Leunis Van Es, 87, of Lincoln, Neb., on August 27 marks the passing of one who truly was a veterinary scientist and one of the most respected veterinarians of the century.

Dr. Van Es, a native of Holland and bearer of academic degrees obtained in three countries (B.S., U. of Amsterdam, 1889; V.S., Ontario V.C., 1893; M.D., U. of Alabama, 1898), had lived on a Nebraska farm for two years, practiced veterinary medicine for two years, and taught histology and bacteriology in a medical school for five years when, at 35, he became head of the Department of Veterinary Science at North Dakota Agricultural College, Fargo, in 1903.

He simultaneously became the state veterinarian and, through his efforts, the Livestock Sanitary Board was soon created. As a result scabies, of both cattle and sheep, and dourine were eradicated from the state, glanders was brought under control, productive research on swamp fever (equine infectious anemia) was initiated, and eradication of bovine tuberculosis was begun.

Also, for several years, starting in 1910, a two-year course of veterinary science was offered under his direction.

In 1918, Dr. Van Es started his 28 years of outstanding service as chairman of the Animal Pathology and Hygiene Department at the University of Nebraska. By 1946, when he relinquished the chairmanship to become professor emeritus, he had produced at least 100 scientific papers, ranging from poisoning to surgery and from anaphylaxis to the identification of stolen meat. Since then, in 1949, he was co-author of an article on swine erysipelas.

"American Men of Science" credits Dr. Van Es with important contributions on "Tetanus; bovine and avian tuberculosis; avian tuberculin test; glanders; hog cholera; swamp fever; parasitic anaphylaxis; immunity to *Bacillus bipolaris;* host relations of *Bacillus tuberculosis;* tuberculosis of swine and other mammals; enzootic liver cirrhosis; epizootiology; anthrax; swine erysipelas; principles of animal hygiene and preventive veterinary medicine," the latter item being the title of his 768-page textbook published in 1932.

These indicate his versatility, but the sterling quality of his keenly analytical mind as well as his uncompromising courage were perhaps best exemplified by the report referred to above as "immunity to *Bacillus bipolaris.*"

The diagnosis of "hemorrhagic septicemia" was well on its way to its unprecedented popularity before Dr. Van Es left North Dakota. His report, in 1920, on the ineffectiveness of the specific vaccine, including the statement that "no reliance can be placed on the vaccines and bacterins against fowl cholera, which . . . are . . . on the market . . ." was attacked by many. In his reply (*J.A.V.M.A.*, Feb., 1921: 572), Dr. Van Es stated that "publication . . . of experiments with commercial vaccines and bacterins against hemorrhagic septicemia and fowl cholera. . .has induced many. . .to assail this work, showing even a little tinge of temper. . . In quasi-veterinary journals and in beautifully decorated advertisements they have undertaken to prove . . . that the scientific foundation for the use of hemorrhagic septicemia vaccines and bacterins can not be questioned."

Apparently his warning voice was smothered by the beautiful advertisements because only now, a third of a century later, does the fad seem to be markedly waning.

From the North Dakota station his followers later reported (*J.A.V.M.A.*, Dec., 1922: 348) that, in extensive experiments, they had failed to infect hogs with Pasteurella; that "the pathogenicity of *Bacterium bipolaris suisepticum* for hogs is purely negligible," that these organisms are "at most, accidental invaders." Yet, even today, official reports from the Agricultural Research Service, U.S.D.A., bear the heading "Part 76—Hog Cholera, *Swine Plague,* and Other Communicable Diseases" (italics supplied), indeed, an exalted position for a disease of such doubtful identity.

Fortunately, more heed was given the findings of Dr. Van Es on most other diseases. His work with swine erysipelas was fully accepted and greatly influenced our present program of control.

Until recent months, Dr. Van Es faithfully submitted, in his distinctive longhand, abstracts from the Dutch veterinary literature. He richly deserved the Twelfth International Congress Prize awarded to him at our Ninetieth Annual Meeting in Toronto in 1953.

*An obituary appears on page 346.

Current Literature

ABSTRACTS

Muscular Dystrophy in Lambs

Blood samples from 128 pregnant ewes and milk samples from 187 ewes fed hay from an area where muscular dystrophy was prevalent and from an area where it did not occur were analyzed for tocopherol. Both a decrease in plasma tocopherol during the last months of pregnancy and a decrease in milk tocopherol during the first days of lactation were observed. There was no significant correlation between either plasma or milk tocopherol levels and the occurrence of muscular dystrophy in the lambs.

Addition of barley, oats, commercial protein supplement, or N, N' -diphenyl-p-phenylenediamine (DPPD) to the hay ration did not alter milk or plasma tocopherol levels. Addition of a commercial protein supplement to the ewes' hay ration almost completely prevented muscular dystrophy in the lambs.—[*J. W. Safford, K. F. Swingle, and D. E. Roberts: Muscular Dystrophy in Lambs as Related to the Tocopherol Levels in the Plasma and Milk of Ewes and to Various Feeds. Am. J. Vet. Res., 17, (July, 1956): 503-508.*]

Diseases of Laboratory Animal Transmissible to Man

The more important diseases of rats, mice, rabbits, birds, dogs, cats, sheep, and monkeys that may be communicable to man are discussed. The hazards in the laboratory are not considered great, but every effort should be made to reduce or eliminate them. If laboratory animals are produced from reliable sources, many of the hazards will not develop. Good management will also prevent hazardous conditions from developing.—[*W. A. Hagan: Diseases of Laboratory Animals Transmissible to Man. Proc. Animal Care Panel (1955): 26-29.*]—N. R. BREWER.

Analyses of Serum from Chickens with Newcastle Disease Virus

White Rock chickens, 3 months old, were inoculated with Newcastle disease virus and were bled 12, 20, and 28 days after infection. Paper electrophoretic analyses of the serum showed a notable decrease in migration of the 12-day sample. This sample also showed an increase in globulin of approximately 20 per cent as determined by Na_2SO_4 precipitation. No increase was discernible on the electropherogram of any globulin fraction.—[*Marcel A. Ginchereau and Harold L. Chute: Electrophoresis Analyses of Serum from Chickens Infected with Newcastle Disease Virus. Am. J. Vet. Res., 17, (July, 1956): 531-534.*]

Experimental Canine Nephritis

An attempt was made to reproduce, experimentally, the lesions of canine interstitial nephritis by the injection of either lithium carmine or *Staphylococcus aureus* toxin directly into the renal artery of dogs. The nephrotoxic agent was injected unilaterally; one kidney was maintained as a control. The animals were surgically prepared by explantation of the urinary bladder to the ventral body wall, allowing the separate collection of urine from the injected and control kidneys.

Functional changes were determined by periodic clinical urinalysis as well as pre- and post-treatment renal clearance trials. Although lower dosage levels (0.1 to 0.18 ml./lb.) of lithium carmine were associated with only an increase in urinary albumin and sediment, a higher dose (0.3 ml./lb.) was accompanied by morphological changes and by a marked decrease in renal clearance. The morphological changes appeared to be indistinguishable from those seen in clinical cases of canine interstitial nephritis. The Staphylococcus toxin appeared to have, predominantly, a necrotizing effect.—[*C. E. Stevens, A. F. Sellers, and J. J. Clark: Studies on Experimental Canine Interstitial Nephritis. II. An Attempt at Functional-Morphological Correlation in Damaged Kidneys. Am. J. Vet. Res., 17, (July, 1956): 389-397.*]

Hydroxycorticosteroid Levels in Sheep and Cattle

Hydrocortisone was administered intravenously to normal cattle and sheep at dosage levels of 0.5 to 1.0 mg. per pound. Clearance of the plasma to normal levels occurred in 6 of 8 ruminating animals within one hour and in the remainder within two hours. The plasma titer of a newborn calf was still markedly elevated at 150 minutes and that of a newborn lamb, although elevated above normal levels at 150 minutes, was approximately half that of the calf. Biliary stasis and early liver fibrosis in a wether resulted in abnormal plasma retention of the steroid.

Recovery of hydrocortisone from urine of 9 animals was 0.18 to 0.77 per cent of the injected dose, indicating that bovine and ovine kidneys can not be considered to be major excretory organs for the elimination of hydrocortisone. Analysis of bile obtained via hepatic duct cannulation of 2 sheep gave a recovery of 0.23 and 1.1 per cent of the injected dose. The total recovery of hydrocortisone from both urine and bile in 1 ewe was 0.49 per cent.—[*Louis W. Holm and Gladys Firch: Plasma, Urine, and Bile Levels of 17-Hydroxycorticosteroids in Sheep and Cattle Following the Administration of Hydrocortisone. Am. J. Vet. Res., 17, (July, 1956): 517-520.*]

BOOKS AND REPORTS

Animal Diseases—The Yearbook of Agriculture, 1956

This yearbook of the U. S. Department of Agriculture is devoted to information about the cause, nature, and prevention of animal diseases.

The preface emphasizes that the book is not a "veterinary handbook" but instead attempts to warn the owner that there is much he can not do

and should not attempt to do for a sick animal. Treatment and cure, if there is a cure, are the responsibility of the veterinarian.

The foreword, written by the Secretary of Agriculture, Ezra Taft Benson, contains the following statement which is indicative of an improved appreciation of the importance of veterinary research and service within the U.S.D.A.: "A challenge of the next 25 years lies in the ability of our colleges to produce scientists with a high specificity of knowledge and a sharpened desire to follow a career devoted to biological and veterinary research. That would suggest a greater specialization in restricted fields of inquiry during the training period. It should not be at the expense of training the general practitioner, but rather in greater selectivity in choosing undergraduates . . . . A part of the problem is that financial opportunities often favor the general practitioner, but the satisfaction of conducting research toward a solution of disease problems is great, and the work of advancing biological knowledge will develop basic facts that in turn will benefit the general practitioner."

The text contains 134 chapters by leading veterinarians and other scientists, most of them in the Department of Agriculture and state colleges.—[*Animal Diseases—The Yearbook of Agriculture, 1956. Superintendent of Documents, Government Printing Office, Washington 25, D. C. 1956. $2.00.*]—H. E. KINGMAN, JR.

Veterinary Obstetrics and Genital Diseases

The text covers the subjects of obstetrics and genital diseases in male and female domestic animals. The author states that "At the outset the author planned a text limited only to obstetrics and genital disease in cattle. . . . As into Noah's Ark, the other animals soon followed. . . . This book was written for veterinary undergraduates and those practitioners who are still students."

The book is large. It does not fit well on the shelf with other books—it is designed to lie on the desk where it can be found and read. It is easy to read. The pages are divided, making the lines short and restful. The type is black and plain. Clear thinking permits the author to use short sentences. He has approached his task modestly, but this was not necessary since such an attitude strengthens one's faith in his opinions. He has given due credit to a long list of others who have worked in these fields. He accepts the risk of too much detail in the belief that many veterinarians want to know everything that can be helpful in the understanding and handling of problems relating to reproduction. There are few tables.

The book is adequately illustrated. Basic subjects are kept at the lowest level compatible with scientific approach. The author might have reduced them still further without detracting from his objectives. Established methods, opinions, and techniques are preserved, and the most recent information on genital diseases of the male and female, and artificial insemination are discussed.

When the call comes to attend a case of dystocia, there is no predicting what will be needed nor what techniques are to be followed until the clinician is "up to his neck" in the problem. Preparation for such emergencies must be made beforehand. This book deserves a place on the desk of every veterinary clinician.—[*Veterinary Obstetrics and Genital Diseases. By Stephen J. Roberts. 551 pages. Published by the author, Cornell University, Ithaca, N. Y. 1956. Price $10.50.*]—H. E. KINGMAN, SR.

Proceedings of the Society of Animal Breeding

This report consists of an analysis of data relating to the most important factors that influenced the success or failure of the artificial insemination program during the years 1951 through 1953.

The president, Sir Thomas Dalling, discussed the role of the veterinary profession in the development of the program.

The papers read were: "The Analysis of Conception Rates and Other Data in Artificial Insemination," "Handling, Transport, and Storage of Bull Semen," "Artificial Breeding and Herd Management in New Zealand," "Work on Breeding Troubles in Dairy Cattle at Cornell University," "Genetic Aspects of Infertility and Infertility in Cattle," and "The Storage of Bull Semen at Low Temperatures."

The authors frankly faced the difficulties associated with artificial insemination and accompanying infertility. The reports are of especial value because of the data in the form of tables and graphs illustrating means and methods of attacking such problems. The papers are made more valuable because of the constructive criticisms and additions contributed by other members of the Society.—[*Proceedings of the Society of Animal Breeding (Division of the British Veterinary Association). Published by the Society. 1955. No price given.*]—H. E. KINGMAN, SR.

Planning Your Animal Hospital

The second edition of this book contains 36 pages of material in addition to that appearing in the first edition issued in 1950 (see JOURNAL, June, 1950: 460). A number of pictures of newer hospitals are shown, together with floor plans and some inside views.

A chapter on "Practice as a Business Venture" has been given the lead position in the revised text. It is an important consideration in planning a hospital; however, the amount of space devoted to a discussion of the economics of small animal practice is too limited to be of much value to the reader.

This second edition does not contain sufficient new material to make it of value to those who have the first edition in their library. It is, however, a book which will certainly be useful to other veterinarians who are planning to build a hospital for small animals.—[*Planning Your Animal Hospital. By Wayne H. Riser. American Animal Hospital Association, 5335 Touhy Avenue, Skokie, Ill. 1956. Price $4.50.*]—H. E. KINGMAN, JR.

THE NEWS

Dr. Jensen Appointed Dean at Colorado Veterinary School

Dr. Rue Jensen, former professor of pathology, was appointed dean of the School of Veterinary Medicine and chief of the animal disease section of the experiment station at Colorado A. & M. College, effective Jan. 1, 1956.

A native of Glenwood, Utah, Dr. Jensen received the B.S. and M.S. degrees from Utah State Agricultural College, the D.V.M. degree from Colorado A. & M. College in 1942, and his Ph.D. from the University of Minnesota in 1954. He has been a member of the Colorado faculty since 1943, having first been on the

Dr. Rue Jensen

faculty at the University of Louisiana for one year. He has been associate dean of the School since February, 1955. One of the profession's most respected pathologists, Dr. Jensen is the author of many important articles. He is a member of Phi Kappa Phi, Sigma Xi, the American College of Veterinary Pathologists, and the AVMA.

Dr. Jensen succeeds Dean Floyd Cross who retired after being associated with the Colorado A. & M. College since 1914 and dean since 1948.

Dr. Holm Appointed Dean at Oklahoma A. & M. College

Dr. Glenn C. Holm, former dean of the School of Agriculture at the North Dakota Agricultural College, at Fargo, has been appointed dean of the School of Veterinary Medicine at Oklahoma A. & M. College, Stillwater.

A native of Shelley, Idaho, where he was born in 1909, Dr. Holm received his first college degree at the University of Idaho, the B.S. in 1932, M.S. in 1933. He then taught bacteriology and milk hygiene at Iowa State College while earning his D.V.M. degree in 1936. Then after two years in practice, he returned to the University of Idaho and, in 1947, became

Dr. Glenn C. Holm

associate director of the Idaho Agricultural Experiment Station.

In 1947, Dr. Holm joined the staff at the North Dakota school and in 1953 was appointed dean, as well as director of the Agricultural Experiment Station. Although an administrator in agricultural departments, he has continuously worked in veterinary medicine and in developing the departments of veterinary science under his jurisdiction.

Dr. Holm has served as chairman of the Division of Veterinary Medicine of the Land-Grant College Association, and for three years has been its administrative advisor for all regional animal disease research in the North Central Region. He is the author of many articles, dealing mostly with pathology or bacteriology. He is also a member of many organizations including Sigma Xi and Phi Zeta.

Dr. and Mrs. Holm will move to Stillwater this fall.

Dr. Olds, Research Fellow, Completes Studies

Dr. Durward Olds (OSU '43) recently completed his graduate studies at the University of Illinois as an AVMA Research fellow. His Ph.D. thesis, "The Composition and Significance of Luminal Fluids in the Bovine Female Genitalia," is on file in the AVMA library.

Dr. Olds was born (1921) and raised near Conneaut, Ohio, where his father, Dr. B. H. Olds (OSU '11), was in practice. For two years after receiving his D.V.M. degree, he served as a bovine artificial insemination technician in Wisconsin. From July, 1946, to September, 1953, he was employed at the University of Ken-

tucky to train inseminators and to do research and teach a course on bovine reproduction. Since then, he has taken graduate work at the University of Illinois, earning his M.S. degree

Dr. Durward Olds

in 1954 and his Ph.D. in June, 1956. He has returned to the University of Kentucky, as professor of dairy science, where he will continue his work with the physiology of reproduction.

He is the author or co-author of 15 previous articles on breeding problems in cattle. Parts of his thesis will be published soon in an AVMA JOURNAL.

Dr. Olds is a member of the AVMA, the American Dairy Science Association, American Association for the Advancement of Science, Phi Zeta, and Sigma Xi. He is married and has two children.

XVIth International Veterinary Congress to Be Held in 1958—Location to Be Decided

Inquiries about the time and place of the next International Veterinary Congress are partly answered as a result of the meeting of the Permanent Committee in Paris on May 19, 1956.

As previously announced (JOURNAL, March 1, p. 269), Argentina withdrew its invitation for the Congress to meet in Buenos Aires in 1957. Austria's national committee, which had also invited the XVIth I.V.C. to meet in Vienna in 1957, advised the Permanent Committee that the time was now too short for them to organize and plan properly for that year and recommended holding it in 1958. This was generally agreed to.

Recent word from the Permanent Committee is to the effect that neither Austria nor Russia (which had indicated indirect interest in being host to the next Congress) would be able to invite it but that Ireland is a possible host country.

Dr. Jac Jansen, general secretary of the Permanent Committee, reported on the healthy state of the Congress fund to which about 25 of the 39 countries represented on the Permanent Committee now contribute regularly. This fund was established in 1949 in order to put financing of the work of the I.V.C. on a sound basis between sessions. Whereas the Permanent Committee previously had had to operate "hand-to-mouth" and even borrow money at times in order to continue its modest staff duties, the Congress fund now enables it to work on a sound budgetary basis. Since the fund was first set up, a modest surplus has been accumulated.

Member countries make annual contributions through their national veterinary associations, paying 1 shilling (14 cents) per member. On this basis, the AVMA has been paying an annual assessment of about $1,500 for the past few years.

At the May 19 meeting in Paris, Dr. Paul D. Delay of California, ARS representative now stationed in Amsterdam, Holland, represented the United States, having been deputized by Dr. W. A. Hagan, U. S. member of the Permanent Committee.

New England V.M.A. to Meet in Connecticut

The twenty-second annual meeting of the New England Veterinary Medical Association will be held in the Hotel Statler, Hartford, Conn., Oct. 7-10, 1956.

Some of the program participants will be: Charles E. Bild, Miami, Fla.; James Archibald and Francis Milne, both of the Ontario Veterinary College, Guelph; James Baker, Cornell University, Ithaca, N. Y.; James Steele, U.S.P.H.S., Atlanta, Ga.; Maurice Shahan, Plum Island, N.Y.; Wayne Plastridge, University of Connecticut, Storrs; Robert Alkire, U.S.D.A., Hartford, Conn.; and a panel on practice management to be given by a member from each of the New England states.

The women's program will include sightseeing in and around Hartford and a program by G. Fox and Company. A cocktail party, banquet, and music with dancing are arranged for the enjoyment of both the veterinarians and their wives.

s/R. E. LARSON, *Publicity chairman.*

U. S. GOVERNMENT

Dr. Omohundro Heads Poultry Disease Eradication Programs.—Dr. Richard E. Omohundro (KSC '37) has been appointed head of poultry disease eradication programs in the Animal Disease Eradication Branch of the Agricultural Research Service, U.S.D.A.

Dr. Omohundro was formerly stationed in Jefferson City, Mo., as veterinarian in charge of U.S.D.A. animal disease eradication activi-

ties in Missouri. His new responsibilities include the development of federal-state cooperative programs for the control and eradication of poultry diseases, and coordination of the activities of poultry diagnostic facilities throughout the country. Under Dr. R. J. Anderson, chief of the Animal Disease Eradication Branch, Dr. Omohundro will serve also as area director of U.S.D.A. livestock disease eradication activities in the North Central States, including Michigan, Illinois, Ohio, and Indiana.

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Dr. McKee Attends Food Hygiene Meeting in Netherlands.—In August, Dr. G. S. McKee (OSU '33), poultry pathologist, Agricultural Marketing Service, attended the meeting of the International Association of Veterinary Food Hygiene in Utrecht, Netherlands, as an official representative of the U. S. Department of Agriculture. During the program, he presented a paper on the causes and detection of poultry diseases, and explained the standards required by the U.S.D.A. Poultry Inspection Service.

AMONG THE STATES AND PROVINCES

Alberta

Alberta Association.—The fifty-first annual convention of the Alberta Veterinary Medical Association was held on June 22-23, 1956, at the Macdonald Hotel in Edmonton.

The following were program participants: E. E. Ballantyne, Edmonton; Floyd Cross, president of the AVMA, Fort Collins, Colo.; K. F. Wells, Ottawa, Ont.; Robert Connell, Lethbridge, Alta.; Morris Erdheim, Chicago, Ill.; J. G. O'Donoghue, Edmonton; L. W. McElroy, Edmonton; T. Lloyd Jones, Guelph, Ont.; and Elmer Clark, Morden, Man.

The entire second morning session was devoted to nutritional problems and the members of the Feed Manufacturers Association were invited to attend. More than 50 feed manufacturers from Alberta were guests of the Association when feed manufacturer and veterinarian relationships were discussed.

s/H. C. CARLSON, *Secretary.*

California

Central Association.—At a recent meeting of the Central California Veterinary Medical Association, the following officers were elected for the 1956-1957 term: Ian C. McDonald, Visalia, president; H. M. Atkinson, Lemoore, vice-president; and R. B. Barsaleau, Visalia, secretary-treasurer.

On Aug. 7, 1956, the Association held its annual laboratory meeting at the state laboratory in Fresno, Calif. The speakers included Al Tietze and R. S. Dickson, both of Bakersfield; Floyd Elliott, Porterville; and G. N. Lukas, Donald Barr, and Paul Carlson, all of Fresno.

s/R. B. BARSALEAU, *Secretary.*

Colorado

Dr. Anderson Accepts Appointment in Minnesota.—Dr. Robert K. Anderson (COL '44), former chief of veterinary public health services for the Department of Health and Hospitals of Denver, has accepted a position on the faculty at the School of Veterinary Medicine, University of Minnesota, St. Paul.

Dr. Anderson has been active in Colorado professional groups, having served as vice-president of the Denver Area V.M.S., as chairman of many committees for both local and state associations, president of the Colorado Public Health Association, a member of the board of directors of the Denver Public Health Council, secretary-treasurer and executive board member of the National Conference of Public Health Veterinarians and, for the past three years, as resident secretary for the AVMA. Dr. Anderson has also been editor of the *Rocky Mountain Veterinarian* for the past six years.

Dr. Murray Camner will serve as temporary editor of the *Rocky Mountain Veterinarian* until a permanent editor is appointed.

Florida

Dr. Sippel to Supervise Laboratory at Kissemmee.—Dr. William L. Sippel (UP '40) resigned, in July, as director of the Animal Disease Laboratory, Tifton, Ga., where he had been for about ten years. He is supervising the building of a new animal disease laboratory at Kissemmee, Fla.

Mississippi

State Association.—The fiftieth annual meeting of the Mississippi State Veterinary Medical Association was held in the Buena Vista Hotel, Biloxi, Miss., on July 15-17, 1956.

Guest speakers and their subjects included the following: C. M. Cooper, Jensen-Salsbery Laboratories, Kansas City, Mo. (bovine mastitis, drug developments); W. S. Gochenour, Pitman-Moore Co., Indianapolis, Ind. (bovine leptospirosis); M. K. Jarvis, Corn States Laboratories, Omaha, Neb. (canine distemper, diseases of the cat); F. H. Oberst, Kansas State College, Manhattan (infertility, large animal problems); and John H. Scruggs, public health service, Atlanta, Ga. (psittacosis in pet birds).

Members of the Association who addressed the group were A. S. Brunton, Vicksburg (canine cough problem); Vernon S. Chadwick, state veterinarian, and L. J. Pate, U.S.D.A., both of Jackson (regulatory work in the state); and Charles H. Horne, Newton, showed a film of pictures taken in practice.

During the business session, the following officers were elected for the ensuing term: George B. Bradshaw, Macon, president; Jack

B. Ross, Jackson, president-elect; James W. Patterson, Columbia, vice-president; and Harvey F. McCrory, State College, secretary-treasurer.

Among the distinguished visitors present was Dr. T. A. Sigler, Greencastle, Ind., past-president of the AVMA (1926-1927) and honorary member of the Mississippi State V.M.A.

s/HARVEY F. MCCRORY, *Secretary.*

Missouri

Death of Dr. Brody.—Many readers of the AVMA JOURNALS will be saddened to learn of the unexpected death of Dr. Samuel Brody (Ph.D.) from coronary thrombosis on Aug. 6, 1956. Dr. Brody, who was professor of dairy husbandry at the University of Missouri, had made many important contributions to the field of animal physiology.

s/H. E. DALE, *University of Missouri.*

New Brunswick

Joint Conference of Maritime Associations.—The seventh annual joint conference of Maritime Veterinary Associations was held at Mount Allison University, Sackville, N. B., June 26-28, 1956. Fifty-eight veterinarians from the four Atlantic provinces and Maine attended.

The program included the following speakers and their subjects: D. C. Maplesden, Ontario Veterinary College, Guelph (antibiotics and sulfonamides, large animal practice); I. W. Moynihan, Animal Diseases Research Institute, Hull, Que. (trichomoniasis, leptospirosis, and vibriosis); R. C. Snyder, Upper Darby, Pa. (ethical procedure, small animal treatment in dairy practice); F. D. Horney, Ontario Veterinary College, Guelph (mucosal disease, bovine practice problems); L. W. MacPherson, Animal Diseases Research Institute, Hull, Que. (respiratory diseases of poultry); H. E. Knapp, Moncton, N. B. (tuberculin and johnin reactors); E. E. Ballantyne, president of the Canada V.M.A., Edmonton, Alta. (disease conditions in Alberta); and E. B. North, Kentville, N. S. (swine diseases).

Dr. Clayton Baxter, Department of Philosophy, Mount Allison University, discussed "The Search for Bridey Murphy" at the banquet which was enjoyed by 103 delegates and their wives.

s/J. F. FRANK, *Resident Secretary.*

North Carolina

Association Approves Fluoridation of Water Supplies.—The North Carolina V.M.A. recently went on record as recognizing that adding 1 p.p.m. of approved fluoride compound to public water supplies, not naturally containing fluorides, would not endanger the health of animals. It encouraged members to actively participate in local efforts to have public water supplies fluoridated.—*Vet. Pub. Health, July, 1956.*

Pennsylvania

State Association.—The seventy-fourth annual convention of the Pennsylvania State Veterinary Medical Association was held on Sept. 11-14, 1956, at the Bedford Springs Hotel, Bedford Springs, Pa.

The following were program participants: Harold S. Bryan, University of Illinois, Urbana (leptospirosis); Jacques Jenny, University of Pennsylvania, Philadelphia (intramedullary pinning techniques); L. H. Bull, deputy secretary of agriculture, Harrisburg (where do we go from here); S. F. Scheidy, Sharpe and Dohme, Philadelphia (new therapeutic agents); Clifford C. Beck, Michigan State University, East Lansing (mycotic mastitis); Myron C. Fincher, Cornell University, Ithaca, N. Y. (infectious mastitis); Lester R. Barto, Basking Ridge, N. J. (canine rectal diseases); Samuel Guss, Pennsylvania State University, University Park (veterinary extension program); Roy F. Davenport, Glenside (dairy herd examinations); Wilson L. Miller, Rohrerstown, Pa. (poultry industry); Stanley Jaks, Zurich, Switzerland (curiosities of the mind); R. L. Elsea, BAI, Harrisburg, Pa. (veterinarian and state laboratory); John W. Fague, Shippensburg, Pa. (bovine restraint); H. E. Nichols, Greenville, Pa. (intravenous fluids, collection of blood); Alan Bachrach, Philadelphia (pet bird treatments); William C. Glenney, Wynnewood, Pa. (renal and cystic calculi); Howard M. Mershon, Linesville, Pa. (dystocia); Raymond E. McKinley, Erie, Pa. (cattle practice); and James V. McCahon, Downingtown, Pa. (blood and intravenous therapy).

s/RAYMOND C. SNYDER, *Secretary.*

Utah

Dr. Boam Featured in Livestock Paper.—Dr. Grant Boam (COL '49), Salt Lake County, was the subject of a two-page article in the *Western Livestock Journal* of Aug. 2, 1956. The article includes a number of excellent pictures showing how a typical large animal practitioner cares for his patients.

s/ARTHUR FREEMAN.

FOREIGN NEWS

France

WHO Public Health Recommendation Adopted.—A new French law provides that specialized veterinary public health studies be included in the curriculums of the veterinary schools. This is in accordance with a recommendation made by the Public Health Advisory Group of the World Health Organization.

In the fourth year of the veterinary curriculum, the student must specialize in one of the following three major areas: animal husbandry and nutrition; animal-processing in-

dustries and inspection; or administration and public health. The choice is left to the student.

Dr. Martin Kaplan, veterinary consultant to WHO, points out that this is a step forward in the teaching of veterinary public health in Europe and believes it will stimulate other nations to take similar action.

s/JAMES H. STEELE, *WHO Consultant.*

Republic of the Philippines

Dr. Aranez Completes Studies in U. S.—Dr. Jose B. Aranez (PHI '46) has received his M.S. degree from Cornell University. Part of his studies were on artificial insemination. He has returned to the College of Veterinary Medicine, University of the Philippines, Quezon City, where he is in charge of the large animal and ambulatory clinics. Dr. Aranez is corresponding secretary for the AVMA.

s/A. D. ALONTE.

VETERINARY MILITARY SERVICE

Short Course in Diseases of Laboratory Animals.—A postgraduate short course on pathology of diseases of laboratory animals is scheduled for Dec. 10-14, 1956, at the Armed Forces Institute of Pathology, Walter Reed Army Medical Center, Washington, D.C. The course is designed to provide training for professional officers who have charge of procurement and maintenance of animal colonies and is intended particularly to help them interpret natural diseases which may influence the supply of laboratory animals or their suitability for experimental use.

For information as to the eligibility requirements, qualifications, application procedures, and selection of students, refer to Circular 621-22, Department of the Army, Washington 25, D. C., dated June 12, 1956.

DEATHS

★**Leunis Van Es** (ONT '93), 87, Lincoln, Neb., died Aug. 27, 1956. Born in Melissant, Holland, Oct. 3, 1868, Dr. Van Es received his early education in that country.

The life story of Dr. Van Es includes his immigration to Nebraska in 1889; graduation (V.S.) from the Ontario Veterinary College in 1893; practicing in Nebraska and in Alabama; earning his M.D. degree at the University of Alabama in 1898; teaching histology and bacteriology at the latter school for five years; heading the Department of Veterinary Science at North Dakota Agricultural College and serving as state veterinarian for 15 years; then, from 1918 to 1946, heading the Department of Animal Pathology and Hygiene at the University of Nebraska (*See* editorial, p. 339).

Dr. Van Es was a member of many professional and scientific societies, including the U.S. Livestock Sanitary Association of which he was president in 1927. An honor roll member of the AVMA since 1950, he had served the Association as second vice-president (1911-1912) and for many years as foreign abstractor

Dr. Leunis Van Es

of Dutch publications. Among the honors bestowed upon him were the honorary degree of Doctor of Science by the University of Pennsylvania (1935); honorary degree of Doctor of Agriculture by the North Dakota Agricultural College, which also dedicated its newly remodeled laboratory in his honor in 1952 and erected a bronze plaque commemorating his work at that institution; and the Twelfth International Veterinary Congress Prize, awarded him by the AVMA in 1953.

Dr. Van Es is survived by two daughters, Mrs. W. C. Zulauf of Pomona, Calif., and Mrs. C. Rumbolz of Lincoln; a son, J. Van Es of Des Moines, Iowa; four grandchildren and five great grandchildren.

• • •

George A. Blohm (ISC '24), Des Moines, Iowa, died June 4, 1956. Dr. Blohm had served with the ARS, U. S. Department of Agriculture.

M. J. Enlow (KCV '13), 76, Pratt, Kan., died May 11, 1956. Dr. Enlow had practiced in Pratt for nearly 40 years. He had been a member of the AVMA.

★**J. M. Newby** (CVC '10), 64, Mount Hamill, Iowa, died July 31, 1956. Dr. Newby had practiced in Mount Hamill for 47 years. He was a member of the AVMA.

Herbert Pew (KCV '06), 78, Niobrara, Neb., died in an automobile accident on June 26, 1956. Dr. Pew had practiced in Niobrara for more than 20 years. He was active in community activities and had served as secretary of the Community Club. His daughter survives.

★Indicates members of the AVMA.

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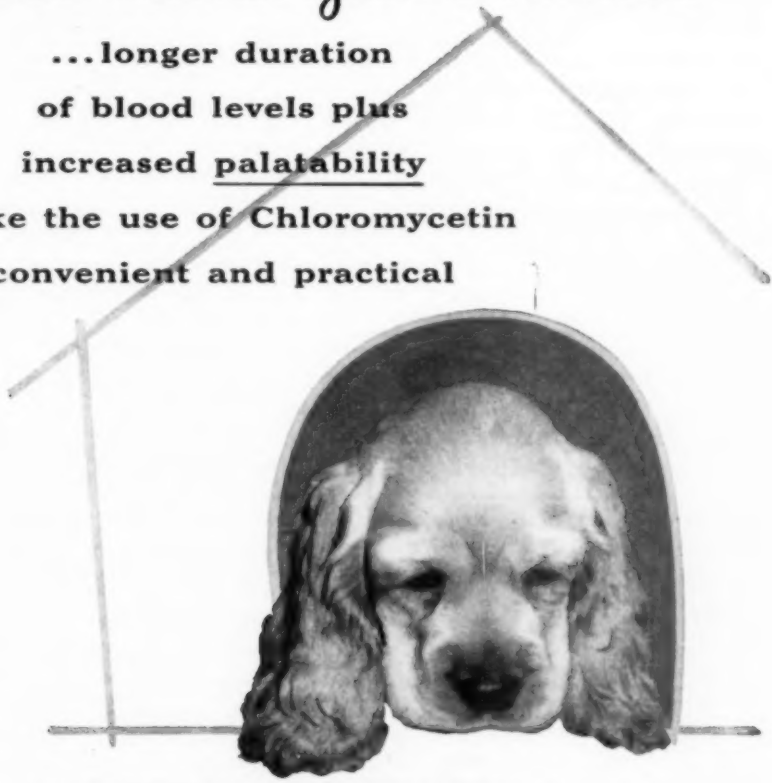

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PROFESSIONAL LITERATURE AVAILABLE ON REQUEST

Department of Veterinary Medicine
PARKE, DAVIS & COMPANY
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ORGANIZATION SECTION

STUDENT CHAPTER ACTIVITIES

Pennsylvania Chapter.—The Women's Auxiliary to the University of Pennsylvania Student Chapter of the AVMA held a banquet May 9 honoring the graduating senior wives.

The program included a talk about the AVMA auxiliaries, given by Mrs. Samuel F. Scheidy, and the conferring of the P.H.T. (putting husband through) degrees by Dean Allam.

s/JANE ENGLISH, *Secretary.*

WOMEN'S AUXILIARY

Acting President—Mrs. Alfred E. Coombs, P.O. Box 174, Skowhegan, Maine

Secretary—Mrs. F. R. Booth, 3920 E. Jackson Blvd., Elkhart, Ind.

Pennsylvania Auxiliary.—The Women's Auxiliary to the Pennsylvania State Veterinary Medical Association held a meeting at the Bedford Springs Hotel, Bedford Springs, Pa., Sept. 11-14, 1956, in conjunction with the seventy-fourth annual convention of the Pennsylvania Veterinary Medical Association.

Aside from the business meeting, the program included a barn dance, a fellowship dinner followed by dancing, and an auction.

s/MRS. B. ZACKON, *Secretary.*

APPLICATIONS

Applicants—Members of Constituent Associations

In accordance with paragraph (b) of Section 2, Article X, of the Administrative Bylaws, as revised at the annual meeting of the House of Representatives, Aug. 18, 1951, in Milwaukee, Wis., the names of applicants residing within the jurisdictional limits of the constituent associations shall be published once in the JOURNAL.

The following applicants have been certified as members of the constituent association that has jurisdiction over the area in which the applicant resides. The certification was made by the secretary of the constituent association in accordance with Section 2, Article X, of the Administrative Bylaws.

ABT, FRANZ
3806 S. Broadway, Los Angeles, Calif.
D.V.M., Zagreb Veterinary College, 1938.

FEDYNIAK, VLADIMIR
916 N. Hoyne Ave., Chicago, Ill.
D.V.M., Hannover Veterinary College, 1950.

FENICHEL, STANLEY H.
43 Virginia Terrace, Red Bank, N.J.
D.V.M., Middlesex University, 1944.

FLAGG, DEAN E.
202 Teton Ave., Bismarck, N. Dak.
D.V.M., Ohio State University, 1945.

FRANKLIN, TED E.
Box 211, College Station, Texas.
D.V.M., Texas A. & M. College, 1941.

GRAVES, FRANCIS M., Jr.
Madison, Va.
D.V.M., Texas A. & M. College, 1955.

HOPSON, BEN W.
1020 Meadow, P.O. Box 1105, Laredo, Texas.
D.V.M., Texas A. & M. College, 1949.

LAMBERT, ROBERT S.
791 E. 332nd St., Eastlake, Ohio.
D.V.M., Ontario Veterinary College, 1944.

MORRIS, ROBERT K.
P. O. Box 757, Lake Charles, La.
D.V.M., Texas A. & M. College, 1948.

POINDEXTER, ALFRED N.
P. O. Box 2591, Prairie View A. & M. College, Prairie View, Texas.
D.V.M., Kansas State College, 1945.

Applicants—Not Members of Constituent Associations

In accordance with paragraph (b) of Section 2, Article X, of the Administrative Bylaws, as revised at the annual meeting of the House of Representatives, Aug. 18, 1951, in Milwaukee, Wis., notice of all applications from applicants residing outside of the jurisdictional limits of the constituent associations, and members of the Armed Forces, shall be published in the JOURNAL for two successive months. The first notice shall give the applicant's full name, school, and year of graduation, post office address, and the names of his endorsers.

First Listing

BARNES, JACK M.
7112 Wayne Ave., Kansas City, Mo.
D.V.M., Texas A. & M. College, 1946.
Vouchers: J. A. Rooker and E. H. Akins.

DAWSON, BERT L.
3510 27th., Lubbock, Texas.
D.V.M., Kansas State Veterinary College, 1914.
Vouchers: J. C. Hart and F. G. Harbaugh.

LEE, CHANG HI
Anyang, National Institute for Veterinary Research
Anyang Kyongki-Do, Korea.
D.V.M., Tokyo Azabu Veterinary College, 1938.
Vouchers: C. E. W. Baker and E. H. Peterson.

PLOCHER, PHILIP D.
Det. 2, Zone 1, 2nd AA Vet SU, Salisbury, Md.
D.V.M., University of California, 1954.
Vouchers: W. H. Horn and E. B. Boroson.

TERRY, JOHN L., JR.
2028 Cascade, Richland, Wash.
V.M.D., University of Pennsylvania, 1944.
Vouchers: L. K. Bustad and L. J. Seigneur.

Second Listing

GODOY, LUIS A. M., Carrera 9a 29-24, Cali, Colombia, S.A.

Graduate Applicants

The following are graduates who have recently received their veterinary degree and who have applied for AVMA membership under the provision granted in the Administrative Bylaws to members in good standing of student chapters. Applications from this year's senior classes not received in time for listing this month will appear in later issues. An asterisk (*) after the name of a school indicates that all of this year's graduates have made application for membership.

First Listing

University of California

CARMICHAEL, LELAND E., D.V.M.
732 Fairview Ave., Arcadia, Calif.
Vouchers: J. F. Christensen and G. D. Pettit.

Michigan State University

McCLUMPHA, CLIFFORD, D.V.M.
831 E. Main St., Owosso, Mich.
Vouchers: C. F. Clark and J. R. DeVries.

University of Minnesota

GRAVES, IRVING L., D.V.M.
South Dakota State College, Department of Veterinary Science, College Station, S. Dak.
Vouchers: D. H. Clifford and R. M. Schwartzman.

SWANSON, RAYMOND B., D.V.M.

2290 Brewster St., St. Paul, Minn.
Vouchers: B. S. Pomeroy and O. H. Osborn.

University of Pennsylvania

FOURNIER, LORRAINE A., V.M.D.
1201 University Ave., New York, N. Y.
Vouchers: E. B. Winokur and C. E. Fletcher.

Texas A. & M. College

BROCK, RALPH H., D.V.M.
700 N. Main, Bryan, Texas.
Vouchers: H. E. Redmond and F. C. Neal.
MASSIE, WINFIELD, D.V.M.
Rt. 1, Midlothian, Va.
Vouchers: W. Bell and I. D. Wilson.

Second Listing

Alabama Polytechnic Institute

JOHNSON, WILLIAM E., D.V.M., Coffee Animal Clinic, LaCenter, Ky.

University of California

ATKINSON, LAWRENCE G., D.V.M., 9966 E. Live Oak, Temple City, Calif.
CALLISON, JACKIE L., D.V.M., P.O. Box 685 Exeter, Calif.
WALKER, LOWELL C., D.V.M., R.F.D. 1, Box 826, Eureka, Calif.

Colorado A. & M. College

BLAKE, ROBERT E., D.V.M., Haystack Angus Ranch, Rt. 2, Longmont, Colo.
BRANDNER, CHARLES E., D.V.M., 206 Welch Ave., Ames, Iowa.
DICKINSON, A. W. III, D.V.M., Rock Springs, Wyo.
HIGHAM, CHARLES W., D.V.M., 610 N. Water Ave., Idaho Falls, Idaho.
KETCHAM, WILLIAM D., D.V.M., 208 W. Prospect, Ft. Collins, Colo.
TAYLOR, MAX E., D.V.M., Show Low, Ariz.
WARD, GILBERT E., D.V.M., 711 N. 22nd St., St. Joseph, Mo.

Michigan State University

ANDERSON, THOMAS P., D.V.M., 324 W. Grand River Ave., Brighton, Mich.
ASBURY, ATWOOD C., D.V.M., School of Veterinary Medicine, University of California, Davis, Calif.
BEITZEL, CAROLINE E., D.V.M., 609 Aspen St., South Milwaukee, Wis.
BELLHORN, ROY W., D.V.M., 6038 Arsenal St., St. Louis, Mo.
BENNE, RICHARD G., D.V.M., 707 S. Lynn, Bryan, Ohio.
BERG, HERMAN, D.V.M., 66 Schley St., Newark, N.J.
BERKMAN, ROBERT N., D.V.M., 312 C. Hickory, East Lansing, Mich.
BILLINGE, RICHARD E., D.V.M., 5996 Massachusetts Ave., Indianapolis, Ind.
BIRR, ROBERT R., D.V.M., 203 M.A.C., East Lansing, Mich.
BRAND, MAX D., D.V.M., Ashley, Ind.
CARTER, ROBERT S., D.V.M., 27460 Bramwell, Farmington, Mich.
CHARTERIS, GERALD C., D.V.M., 288 E. Bennett, Ferndale, Mich.
CLARK, RICHARD D., D.V.M., R.R. 2, Middleville, Mich.
CLINTON, THOMAS M., D.V.M., 12345 Flanders, Detroit, Mich.
CORSON, ORAL D., D.V.M., 5408 Gratiot Ave., St. Clair, Mich.
CRAMTON, KENNETH G., D.V.M., St. Charles, Mich.
DE PORRE, PIERRE L., D.V.M., 21441 Sloan Dr., Harper Woods, Mich.
DOUGLAS, KENDRIC C., D.V.M., 1034 W. Giles Rd., Muskegon, Mich.
DROBISH, EDWARD J., D.V.M., 235 Stoddard Ave., East Lansing, Mich.
EAMES, EDWARD N., D.V.M., 286 Pleasent St., Concord, N. H.
ELKINS, ROBERT W., D.V.M., 717 W. Hamlin Rd., R.F.D. 4, Rochester, Mich.
EPPERT, JOHN W. Jr., D.V.M., R.R. 1, Brazil, Ind.
EWING, DEAN E., D.V.M., 2121 Kenwood, Fort Wayne, Ind.
GROUNDS, F. ORAL, Jr., D.V.M., 3217 Manley Dr., Lansing, Mich.
HERSON, FRANK S., D.V.M., 41 Glen Rd., Winchester, Mass.
HUFF, RICHARD W., D.V.M., 16549 Parkside, Detroit, Mich.
KUDLA, STEVEN A., D.V.M., 821 E. Carton St., Flint, Mich.
KURILCHIK, RICHARD H., D.V.M., 2302 Second St., Wyandotte, Mich.
LIPTAK, ERNEST A., D.V.M., Sycamore Farm, Yardville, N. J.
MURRAY, ALEX P., D.V.M., Box 205, West DePere, Wis.
NAKAGAWA, EDWARD K., D.V.M., 1431 River St., Honolulu, T. H.
NEIDLINGER, GLEN E., D.V.M., 314 Roosevelt, Walkerton, Ind.
OLDT, CHARLES C., D.V.M., 1602 Francis Ave., S. E., Grand Rapids, Mich.
PUTNAM, WILLIAM B., D.V.M., 32 Main Circle, Shrewsbury, Mass.
ROOKS, JOHN F., D.V.M., 1158 Nixon, N. W., Grand Rapids, Mich.
RUFF, WILLIAM R., D.V.M., Whiting Village, R. 1, Stevens Point, Wis.
SASSU, GEORGE, D.V.M., 5518 W. Schubert Ave., Chicago, Ill.
SHIPMAN, DAVID E., D.V.M., 2626 Geddes Ave., Ann Arbor, Mich.
TESSMAN, HOWARD L., D.V.M., 455 N. Center Rd., Saginaw, Mich.
TRAPP, ALLAN L., D.V.M., 128½ W. Sycamore St., Mason, Mich.
UDENANS, MIRDZA, D.V.M., 13492 Sparling, Detroit, Mich.
VANGIESON, VAL C., D.V.M., 50485 Pontiac Trail, Wixom, Mich.
WIELAND, ROGER E., D.V.M., 921-D Walnut Lane, East Lansing, Mich.
WUNDERLICH, CHARLES C., D.V.M., 1901 Wilmington Ave., New Castle, Penn.
YANCHYSHYN, IVAN, D.V.M., 411-D Willow Lane, East Lansing, Mich.

University of Minnesota

HOLLAND, EDWARD R., D.V.M., Barron, Wis.
KARLI, VERNON A., D.V.M., 2079-D Hoyt Ave. W., St. Paul, Minn.
KRUEGER, RUTH H., D.V.M., 5335 Touhy Ave., Skokie, Ill.
SWACINA, DOUGLAS C., D.V.M., 2269 Brewster St., St. Paul, Minn.
WILLIAMSON, JOHN L., D.V.M., 8208 Carnegie Ave., Cleveland, Ohio.

University of Missouri

CLARK, WILLIAM D., D.V.M., 925 N. Lake St., Aurora, Ill.

Ohio State University*

KRANER, KEITH L., D.V.M., Mounted Rt., Rana Villa Ave., Camp Hill, Pa.

University of Pennsylvania

CONSTANTINI, HELENA J., V.M.D., 2437 Overlook Rd., Cleveland, Ohio.
COOK, RICHARD O., V.M.D., Jarrettsville, Md.
FRANKO, GEORGE A., V.M.D., R.D. 1, Falls, Pa.
GARVIN, CHARLES H., V.M.D., 347 Lakeview Ave., Drexel Hill, Pa.
REYNOLDS, HARRY A., D.V.M., 1210 Burlington Ave., Delanco, N.J.

(*Continued on p. 24*)

(APPLICATIONS—*continued from p. 23*)

RYAN, RICHARD R., V.M.D., 6461 Woodcrest Ave., Philadelphia, Pa.

SIMMONS, JAY J., V.M.D., 232 W. Main St., Norristown, Pa.

VERNIMB, GEORGE D., V.M.D., R.F.D. 1, Randolph, Vt.

WHITNEY, ARTHUR B., V.M.D., 1124 Lowell Ave., Cornwells Heights, Pa.

Texas A. & M. College

COX, LAWRENCE F., D.V.M., Border Animal Hospital, Weslaco, Texas.

DISMUKES, HENRY C., Jr., D.V.M., 6604 Bayway Drive, Baytown, Texas.

GUILES, DERRELL H., D.V.M., Box 1239, c/o SAG Services, Dhahran, Saudi Arabia.

KYLE, JAMES W., D.V.M., P.O. Box 568, Kilgore, Texas.

MICHELITCH, EDWARD, D.V.M., R.F.D. 2, Box 555, McLean, Va.

ROE, WALTER E., Jr., D.V.M., Round Hill, Virginia.

TRIMMIER, BILLY R., D.V.M., 435 Shannon Lee, San Antonio, Texas.

WATKINS, JOHN R., D.V.M., 706 Edgemore, Bryan, Texas.

WEST, JOE E., D.V.M., 5335 Touhy Ave., Skokie, Ill.

Tuskegee Institute

NORRIS, AUSTIN C., D.V.M., 511 River Dr., East Paterson, N.J.

Evaluating New Drugs

Commenting on a criticism of an unfavorable report on a controlled experiment with cortisone for arthritis, a correspondent wrote in the *British Medical Journal* (July 7, 1956):

> In the face of a stream of new drugs backed by compelling advertisements, we must keep our feet on the ground and not allow ourselves to be carried away by impressions that some patients have done well because they received this drug and some have done well because they have received that.

This is "Champ," the 18-foot long, 9.25-foot high, plastic Hereford steer produced by the Ralston Purina Company for exhibition over the country. Champ is mounted on a trailer 34 feet long and 8 feet wide for highway traffic but when on exhibition, as shown here, the steer expands to over 11 feet wide so that visitors can walk between the viscera and the body wall. Champ is actually an extreme hemaphrodite—all steer on the left side, all pregnant cow on the right. Except for necessary artistic liberties "he" is built true to life.

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**Jones, S. V.; Belloff, G. B., and Roberts, H. D. B.: Vet. Med. In Press.*

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COMING MEETINGS

Missouri, University of. Annual short course for veterinarians. School of Veterinary Medicine, University of Missouri, Columbia, Oct. 1-2, 1956. Cecil Elder, chairman, short course committee.

Southwestern Iowa Veterinary Medical Association. Semi-annual meeting. Chieftan Hotel, Council Bluffs, Iowa, Oct. 2, 1956. Fred W. Petersen, Avoca, secretary.

Purdue University. Annual short course for veterinarians. Purdue University, Lafayette, Ind., Oct. 3-5, 1956. L. M. Hutchings, Purdue University, Department of Veterinary Science, Lafayette, chairman.

Eastern Iowa Veterinary Association, Inc. Annual meeting. Hotel Montrose, Cedar Rapids, Oct. 4-5, 1956. Forrest E. Brutsman, Traer, secretary.

South Dakota Veterinary Medical Association. Annual meeting. Hotel Cataract, Sioux Falls, Oct. 4-5, 1956. J. L. Noordsy, Marion, S. Dak., secretary.

New England Veterinary Medical Association. Annual meeting. Hotel Statler, Hartford, Conn., Oct. 7-10, 1956. C. Lawrence Blakely, 484 Chestnut St., Needham, Mass., secretary.

University of Illinois. Annual Conference and extension short course for veterinarians. College of Veterinary Medicine, University of Illinois, Urbana, Oct. 12, 1956, L. E. Boley, Department of Veterinary Clinical Medicine, chairman.

American Association of Veterinary Bacteriologists. Annual meeting. Texas A. & M. College, College Station, Oct. 13, 1956. J. P. Delaphane, Michigan State University, College of Veterinary Medicine, chairman.

American Veterinary Medical Association. Annual meeting. Municipal Auditorium, San Antonio, Texas, Oct. 15-18, 1956. J. G. Hardenbergh, 600 S. Michigan Ave., Chicago 5, Ill., executive secretary.

Veterinary Symposium on Dogs. Kankakee Civic Auditorium, Kankakee, Ill., Oct. 24, 1956. Mr. Harry Miller, Gaines Dog Research Center, 250 Park Ave., New York 17, N. Y., director.

Mississippi Valley Veterinary Medical Association. Annual meeting. Hotel Pere Marquette, Peoria, Ill., Nov. 7-8, 1956. William L. Beer, 612 N. College Ave., Aledo, Ill., secretary.

Midwest Small Animal Association, annual meeting, and **American Animal Hospital Association,** regional meeting. Hotel Burlington, Burlington, Iowa, Nov. 18-19, 1956. J. Porter Coble, 2828 S. MacArthur Blvd., Springfield Ill., secretary, Midwest Small Animal Association.

U. S. Livestock Sanitary Association. Annual meeting. Morrison Hotel, Chicago, Ill., Nov. 28-30, 1956. R. A. Headershott, 33 Oak Lane, Trenton 8, N. J., secretary.

Animal Care Panel. Annual meeting. Morrison Hotel, Chicago, Ill., Nov. 29-30, 1956. Robert J. Flynn, P.O. Box 299, Lemont, Ill., secretary.

Nebraska Veterinary Medical Association. Annual meeting. Hotel Lincoln, Lincoln, Dec. 3-5, 1956. W. T. Spencer, 1250 North 37th St., Lincoln, secretary.

New York State Veterinary College. Annual conference for veterinarians. New York State Veterinary College, Cornell University, Ithaca, Jan. 2-4, 1957, W. A. Hagan, dean.

Iowa Veterinary Medical Association. Annual meeting. Hotel Fort Des Moines, Iowa, Jan. 22-24, 1957. F. B. Young, Waukee, Iowa, secretary.

Minnesota State Veterinary Medical Association. Annual meeting. Radisson Hotel, Minneapolis, Feb. 4-6, 1957. B. S. Pomeroy, 1443 Raymond Ave., St. Paul 8, secretary.

Foreign Meetings

Asociación Médico Veterinaria de Puerto Rico. Inter American veterinary medical symposium. Caribe Hilton Hotel, San Juan, Nov. 30-Dec. 2, 1956. O. A. López-Pancheco, P.O. Box 155, Hato Rey, Puerto Rico, chairman.

(Continued on p. 28)

New Instant Dog Food Mix

Absorbs liquid almost instantly, stays crumbly-moist, won't pack. Meaty aroma tempts finicky appetites... 16 health-giving benefits help make dogs champions.

QUICK MIXING meal has everything professional men want

Golden-brown, irregular-shaped kernels of new Quick Ken-L-Meal absorb liquid far faster than any other brand. Stays crumbly-moist, won't pack, won't stick to the bottom of the feeding dish or the roof of your dog's mouth. He can easily eat it all!

MEATY AROMA AND FLAVOR PLUS SUPER NOURISHMENT

High-meat-meal, super nourishing Quick Ken-L-Meal releases all the health-giving benefits of 16 rich, wholesome nutrients into your dog's system—every vitamin and mineral a dog is known to need, plus *even more protein nourishment than fresh-ground meat.*

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To introduce you to the revolutionary benefits of New Quick Ken-L-Meal . . . to let you see for yourself how dogs prefer it to other brands . . . we'll deliver a *free* trial supply so you can test it against your present brand. There is no obligation.

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I want to try new Quick Ken-L-Meal in my kennel. *I want to see how it performs against my present brand of dog food. I am under no obligation whatsoever.*

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*Address*_____ *No. of dogs*_____

*City*_____ *Zone*_____ *State*_____

This offer is limited. So mail this coupon today!

(COMING MEETINGS—*continued from p. 26*)

Regularly Scheduled Meetings

ALABAMA—Central Alabama Veterinary Association, the first Thursday of each month. B. M. Lauderdale, Montgomery, secretary.

Jefferson County Veterinary Medical Association, the second Thursday of each month. S. A. Price, 213 N. 15th St., Birmingham, secretary.

ARIZONA—Central Arizona Veterinary Medical Association, the second Tuesday of each month. Keith T. Maddy, Phoenix, Ariz., secretary.

Pima County Veterinary Medical Association, the third Wednesday of each month in Tucson. E. T. Anderson, 8420 Tanque Verde Rd., Tucson, Ariz., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. E. T. Anderson, Rt. 2, Box 697, Tucson, Ariz., secretary.

CALIFORNIA—Bay Counties Veterinary Medical Association, the second Tuesday of each month. E. Paul, Redwood City, Calif., secretary.

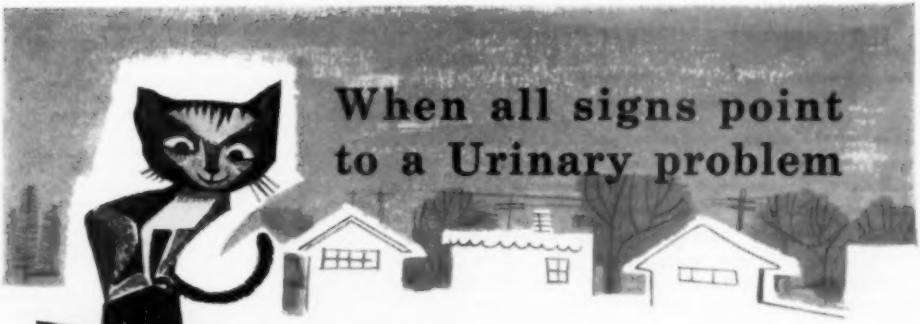
Central California Veterinary Medical Association, the fourth Tuesday of each month. Wilfred Pimentel, 3455 S. Elm Ave., Fresno, Calif., secretary.

Association of East Bay Veterinarians, bimonthly, the fourth Wednesday. Leo Goldston, 3793 Broadway, Oakland 11, Calif., secretary.

Kern County Veterinary Medical Association, the first Thursday evening of each month. A. L. Irwin, 301 Taft Highway, Bakersfield, Calif., secretary.

Mid-Coast Veterinary Medical Association, the first Thursday of every even month. W. H. Rockey, P. O. Box 121, San Luis Obispo, Calif., secretary.

(*Continued on p. 30*)



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Methenamine, salol, methylene blue and benzoic acid combat urinary tract invaders—reduce bacterial and pus-cell content, promote healing.

URISED...

Atropine, hyoscyamine and gelsemium relax smooth muscle along the urinary tract—relieve painful spasm quickly.

URISED...

Unexcelled for long-term therapy—no contraindications, no reports of toxic effects.

URISED...

Formation of alkaline calculi discouraged in acid urine.

To treat all urinary problems of small animals, specify **URISED,** *veterinary*

Sold to Graduate Veterinarians only

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Chicago, Illinois

Pacific Coast Branch
381 Eleventh St.
San Francisco, Calif.

Send for literature and clinical trial supply of **URISED,** *veterinary*

(COMING MEETINGS—*continued from p. 28*)

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. Lewis J. Campbell, 90 Corral de Tierra, Salinas, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Covell, in Modesto, Calif. Lyle A. Baker, Turlock, Calif., secretary.

Orange Belt Veterinary Medical Association, the second Monday of each month. Chester A. Maeda, 766 E. Highland Ave., San Bernardino, Calif., secretary.

Orange County Veterinary Medical Association, the third Thursday of each month. Donald E. Lind, 2643 N. Main St., Santa Ana, Calif., secretary.

Peninsula Veterinary Medical Association, the third Monday of each month. T. D. Harris, San Mateo, Calif., secretary.

Redwood Empire Veterinary Medical Association, the third Thursday of each month. Robert E. Clark, Napa, Calif., secretary.

Sacramento Valley Veterinary Medical Association, the second Wednesday of each month. W. E. Steinmetz, 4227 Freeport Blvd., Sacramento, Calif., secretary.

San Diego County Veterinary Medical Association, the fourth Tuesday of each month. H. R. Rossoll, 1795 Moore St., San Diego, Calif., secretary.

San Fernando Valley Veterinary Medical Association, the second Friday of each month at the Casa Escobar Restaurant in Studio City. John Chudacoff, 7912 Sepulveda Blvd., Van Nuys, secretary.

Southern California Veterinary Medical Association, the last Wednesday of each month. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

Tulare County Veterinarians, the second Thursday of each month. R. B. Barsalesu, 2333 E. Mineral King, Visalia, Calif., secretary.

COLORADO—Denver Area Veterinary Society, the fourth Tuesday of every month. Richard C. Tolley, 5060 S. Broadway St., Englewood, Colo., secretary.

Northern Colorado Veterinary Medical Society, the first Monday of each month. M. A. Hammarlund, School of Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. E. J. Hathaway, Clifton Park Manor, Apt. 73-5, Wilmington 2, Del., secretary.

FLORIDA—Central Florida Veterinary Medical Association, the second Friday of each month, time and place specified monthly. James B. Murphy, Eustis, Fla., secretary.

Jacksonville Veterinary Medical Association, the second Thursday of each month, time and place specified monthly. George F. Yopp, 4644 Main St., Jacksonville, Fla., secretary.

Palm Beach Veterinary Society, the last Thursday of each month in the county office building at 810 Datura St., West Palm Beach. Ross E. Evans, 5215 S. Dixie Highway, West Palm Beach, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. Paul J. Myers, Winter Haven, Fla., secretary.

South Florida Veterinary Society, the third Tuesday of each month, at the Seven Seas Restaurant, Miami, Fla. E. D. Stoddard, 6432 S. W. 8th St., Miami, Fla., secretary.

Suwannee Valley Veterinary Association, the third Friday of each month, at the Thomas Hotel, Gainesville, Fla. R. C. Mann, Rt. 1, Box 37, Ocala, Fla., secretary.

GEORGIA—Atlanta Veterinary Society, the second Tuesday of every month at the Elks Home on Peachtree St., Atlanta, Ga. J. L. Christopher, Smyrna, Ga., secretary.

ILLINOIS—Chicago Veterinary Medical Association, the second Tuesday of each month. Mark E. Davenport, Jr., 215 S. Edgewood Ave., LaGrange, Ill., secretary.

Eastern Illinois Veterinary Medical Association, the first Thursday of March, June, September, and December. A one-day clinic is held in May. H. S. Bryan, College of Veterinary Medicine, University of Illinois, Urbana, secretary.

INDIANA—Central Indiana Veterinary Medical Association, the second Wednesday of each month. Peter Johnson, Jr., 4410 N. Keystone Ave., Indianapolis 5, secretary.

Michiana Veterinary Medical Association, the second Thursday of every month, except July and December, at the Hotel LaSalle, South Bend, Ind. J. M. Carter, 3421 S. Main St., Elkhart, Ind., secretary.

Tenth District Veterinary Medical Association the third Thursday of each month. W. E. Sharp, Union City, Ind., secretary.

IOWA—Cedar Valley Veterinary Association, the second Monday of each month, except January, July, August, and October, at Black's Tea Room, Waterloo, Iowa. H. V. Henderson, Reinbeck, Iowa, secretary.

Coon Valley Veterinary Association, the second Wednesday of each month, September through May, at the Bradford Hotel, Storm Lake, Iowa. D. I. Lee, Sac City, Iowa, secretary.

Fayette County Veterinary Association, the third Tuesday of each month, except in July and August, at Pa and Ma's Restaurant, West Union, Iowa. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Winneslick Hotel, Decorah, Iowa, 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

(*Continued on p. 32*)

for mastitis and topical application

7.5 cc. tube contains:

Neomycin sulfate . . . . . 100 mg.
(equivalent to 60 mg. standard neomycin base)

Polymyxin B sulfate . . . 100,000 Units

in a free-flowing milk miscible base.

100 cc. multi-dose vials, each 10 cc. contains:
Neomycin sulfate . . . . . 100 mg.
Polymyxin B sulfate . . . 100,000 Units
in a free-flowing milk miscible base

A synergistic antibiotic combination

Daribiotic*

Neomycin—Polymyxin B, Massengill

for intestinal and uterine infections

Each green oblong tablet contains:

Neomycin sulfate. . . . . 25 mg.
(equivalent to 15 mg. standard neomycin base)

Polymyxin B sulfate . . . . . 25,000 Units

conveniently packaged for dispensing in plio-film strips.

Twenty-four individually sealed tablets per carton.

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VETERINARY DIVISION

Bristol, Tennessee

Kansas City • San Francisco • New York

*U.S. Pat. 2,565,057

(COMING MEETINGS—*continued from p. 30*)

KENTUCKY—Central Kentucky Veterinary Medical Association, the first Wednesday of each month. L. S. Shirrell, Versailles Rd., Frankfort, secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday evening of each month in Louisville or within a radius of 50 miles. W. E. Bewley, P.O. Box "H," Crestwood, secretary.

MARYLAND—Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m. at the Park Plaza Hotel, Charles and Madison St., Baltimore, Md. Harry L. Schultz, Jr., 9011 Harford Rd., Baltimore, Md., secretary.

MICHIGAN—Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert E. Kader, 5034 Armstrong Rd., Lansing 17, Mich., secretary.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. S. Correll, Rt. 1, Midland, Mich., secretary.

Southeastern Veterinary Medical Association, the fourth Wednesday of every month, September through May. Gilbert Meyer, 14003 E. Seven Mile Rd., Detroit 5, Mich., secretary.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of the month (except July and August) at the Sheraton Hotel, Spring Ave. and Lindell Blvd. Allen B. Shopmaker, 136 N. Meramec, Clayton 5, Mo., secretary.

Kansas City Small Animal Hospital Association, the first Monday of each month, at alternating hospitals. W. F. Noland, 7504 Metcalf, Overland Park, Kan., secretary.

Kansas City Veterinary Medical Association, the third Tuesday of each month at Exchange Hall, ninth floor, Livestock Exchange Bldg., 1600 Genessee St., Kansas City, Mo. Busch Meredith, 800 Woodswether Rd., Kansas City 5, Mo., secretary.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hights Inn, Highstown, N. J. David C. Tudor, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April at the Academy of Medicine, 91 Lincoln Park South, Newark, N. J. Myron S. Arlein, 2172 Milburn Ave., Maplewood, N. J., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Casa Mana in Teaneck. James R. Tanzola, Upper Saddle River, secretary.

Southern New Jersey Veterinary Medical Association, the third Tuesday of each month at the Collingswood Veterinary Hospital, Collingswood. W. E. Snyder, E. Kings Highway and Munn Ave., Haddonfield, secretary.

NEW YORK—New York City, Inc., Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel in Greensboro. J. W. Peace, High Point, secretary.

Eastern North Carolina Veterinary Medical Association, the first Friday of each month. Wm. Allen Potts, 401 W. James St., Mount Olive, secretary.

Piedmont Veterinary Medical Association, the last Friday of each month at 7:00 p.m. in Mull's Motel in Hickory.

(*Continued on p. 34*)

(COMING MEETINGS—*continued from p. 32*)

N. Car. W. W. Dickson, Box 1071, Gastonia, N. Car., secretary.

Redmont Veterinary Medical Association, the last Tuesday of the month. J. L. Innes, 9 Tampa Ave., West Asheville, N. Car., secretary.

OHIO—Cuyahoga County Veterinary Medical Association, the first Wednesday of each month, September through May (except January), at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. Ed. R. Jacobs, 5522 Pearl Rd., Cleveland, Ohio, secretary.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month. James M. Brown, 2818 W. Britton Rd., Oklahoma City, secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month in Directors' Parlor of the Brookside State Bank, Tulsa, Okla. Don L. Hohmann, 538 S. Madison St., Tulsa, Okla., secretary.

PENNSYLVANIA—Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania School of Veterinary Medicine, 39th and Woodland Ave., Philadelphia 4, Pa. Raymond C. Snyder, 39th and Woodland Ave., Philadelphia 4, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. J. Marvin Prewitt, 4141 Lexington Blvd., Corpus Christi, Texas, secretary.

VIRGINIA—Central Virginia Veterinarians' Association, the third Thursday of each month at the William Byrd Hotel in Richmond at 8:00 p.m. M. R. Levy, 312 W. Cary St., Richmond 20, Va., secretary.

Northern Virginia Veterinary Society, the second Wednesday of every third month. Meeting place announced by letter. H. C. Newman, Box 145, Merrifield, Va., secretary.

Southwest Virginia Veterinary Medical Association, the first Thursday of each month. I. D. Wilson, Blacksburg, secretary.

WASHINGTON—Seattle Veterinary Medical Association, the third Tuesday of each month in the Trinity Episcopal Church, 8th and James St., Seattle, Wash. P. R. Des Rosiers, 5508 2nd Ave., N. W., Seattle 7, Wash., secretary.

South Puget Sound Veterinary Association, the second Thursday of each month except July and August. O. L. Bailey, P. O. Box 906, Olympia, Wash., secretary.

WEST VIRGINIA—Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the second Thursday of each month in the Hotel Prichard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W., Huntington, W. Va., secretary.

WISCONSIN—Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. George F. Lynch, 201 West Devon St., Milwaukee 17, Wis., secretary.

Dr. Sanders Joins Pitman-Moore Staff

Dr. E. F. Sanders has been named to head Pitman-Moore Company's tissue product department, according to Dr. S. R. Bozeman, director of the firm's biological laboratories.

Dr. Sanders received his D.V.M. degree from Kansas State College. In 1931, he became affiliated with Jensen-Salsbery Laboratories, Inc., Kansas City, and resigned as vice-president and director of the biological division to join the Pitman-Moore biological staff.

PARLAMATE

piperazine citrate, PARLAM

eliminates WORMS in one treatment

'PARLAMATE' IS IDEAL for LARGE and SMALL ANIMAL USE

Safe New All-Species, Anthelmintic up to 85-100% Effective Against Roundworms and Nodular Worms

- Safe, fast and most effective.
- Non-toxic and easily tolerated.
- No hair or feather staining, hepatic damage, gastro-intestinal irritation, or other serious side effects.
- No pretreatment starving or purging necessary.
- Very palatable.
- Easily administered in feed or drinking water, or by dose syringe, drench or stomach tube.
- Only 1 treatment needed.
- 85-100% effective against mature and immature large roundworms (Ascaris) and nodular worms (Oesophagostomum).
- Long acting durable effect; parasites passed for several days.
- Economical to use.

DOSAGE FORMS

'PARLAMATE' Powder (contains 100% piperazine citrate). In preweighed dispensing packages convenient for office and field use.
Bottles of 1 oz., 4 ozs., 1 lb.

'PARLAMATE' Tablets, 250 mg. (each tablet contains the equivalent of 100 mg. available piperazine).
Bottles of 100 and 1,000 tablets.

'PARLAMATE' Liquid. (Each teaspoonful, approximately 5 cc., supplies 250 mg. piperazine citrate, which is equivalent to 100 mg. available piperazine.)
Bottles of 1 pint and 1 gallon.

'PARLAMATE' IS NOW AVAILABLE FOR IMMEDIATE DELIVERY

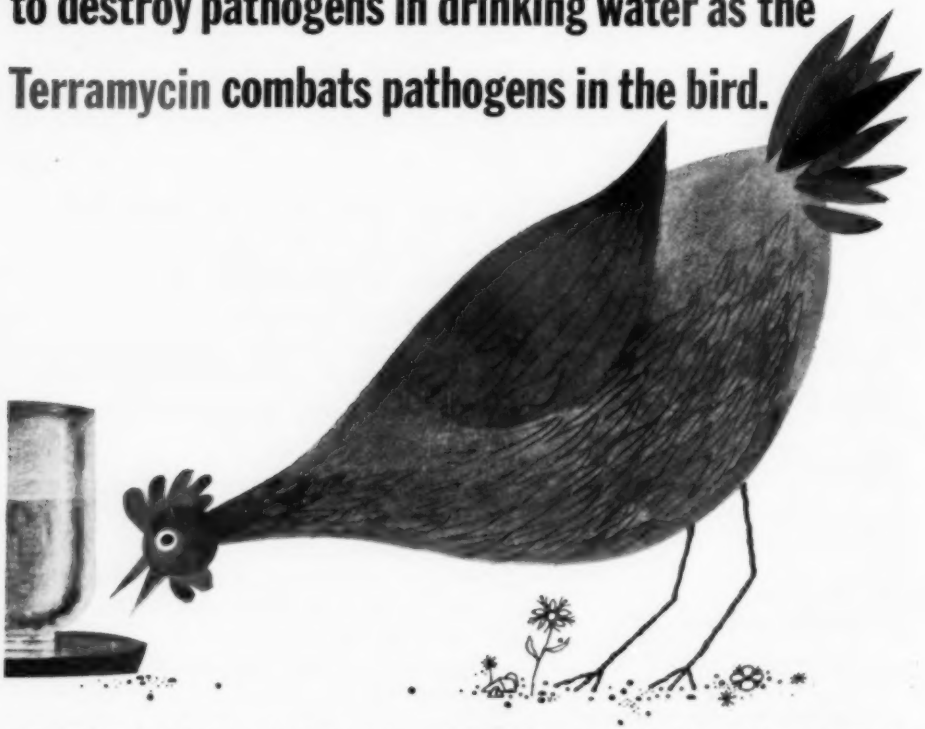
At Your Supplier, or Write

Parlam CORPORATION • 340 Canal Street, New York 13, N. Y.

"Devoted to progressive veterinary research for the development of superior products for graduate veterinarians."

Sole Canadian Distributor: Veterinary Medical Supply Co. of Canada, 6579 Somerled Ave., Montreal, Que.

Now better than ever! Terramycin®* Poultry Formula now contains Anti-germ 77† (benzethonium chloride) to destroy pathogens in drinking water as the Terramycin combats pathogens in the bird.



ANTI-GERM 77 disinfects the drinking water to prevent the water-borne spread of fowl cholera, fowl typhoid, pullorum, paratyphoid, infectious diarrhea, enteritis, and other bacterial infections.

TERRAMYCIN exerts its well-proved broad-spectrum effect in the prevention, treatment, or management of blue comb, CRD, hexamitiasis, and infectious synovitis, and in the stimulation of healthy growth and egg production.

Terramycin Poultry Formula

Each pound represents 25 Gm. of oxytetracycline hydrochloride activity and 25 Gm. of benzethonium chloride (iso-octyl phenoxyethoxyethyl dimethyl benzyl ammonium chloride). Available in ¼ lb., 1 lb., and 5 lb. jars. ***Highly concentrated... completely soluble.***

* Brand of oxytetracycline
† Trademark

Pfizer

Department of Veterinary Medicine, PFIZER LABORATORIES, *Division, Chas. Pfizer & Co., Inc.*, Brooklyn 6, N.Y.

Every Nutrient A Cat Is Known To Need

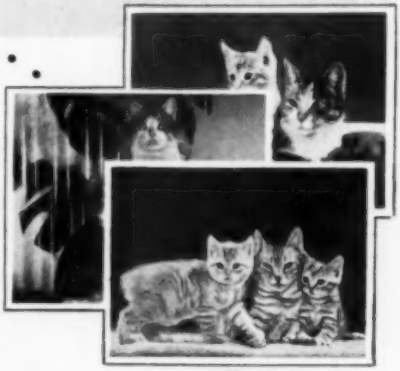
Many prepared cat foods—including some labelled "all fish"—will not supply balanced nutrition without supplementary feeding. Puss 'n Boots Cat Food is scientifically formulated to provide *every* nutrient a cat is known to need. The formula includes:

- ***Whole Fish*** (not scraps or by-products), with nature's own life balance of proteins, vitamins, minerals and other nutrients. Puss 'n Boots does not sell the fish fillets for human consumption, or the liver and glands (rich in vitamins and minerals) for medicinal use. All is retained, even the bone structure for its calcium — after being made soft and digestible.
- ***Selected Cereals,*** added to the finely ground whole fish to provide carbohydrates and roughage for all-round nutrition.
- ***Extra Vitamin B_1,*** added to every can as a margin of safety.

You can recommend Puss 'n Boots to cat owning clients with complete confidence in its nutritional excellence.

FREE! ***For your office walls***

Three cat photographs — in full color, 8" x 10", by Walter Chandoha, world's greatest cat photographer. Sent to you without charge on receipt of request on professional letterhead. Address Puss 'n Boots, Dept. M-66, Box 6586, Chicago 77, Illinois.



PUSS 'n BOOTS is good nutrition

America's largest selling cat food...adds the plus in health, beauty, vigor.

Coast Fisheries, Division of The Quaker Oats Company, Chicago 54, Ill.

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Personal Want Ads—$4.00 for the first 25 words and 10 cents for each additional word; 35 cents for use of box number.

Commercial Want Ads—$5.00 for the first 25 words, 25 cents for each additional word.

Remittance must accompany ad.

Deadlines for Want Ads

***For JOURNAL dated 1st of month* — 8th of month preceding date of issue.**

***For JOURNAL dated 15th of month* — 22nd of month preceding date of issue.**

Names of classified advertisers using key letters can not be supplied. Address your reply to the key letters, c/o JOURNAL of the AVMA, 600 S. Michigan Ave., Chicago 5, Ill., and it will be transmitted to the advertiser.

Wanted—Veterinarians

There are vacancies for veterinarians in regulatory work in Logan and Brigham City, Utah. Annual salary, $5,200 and up. Address "Box S 8," c/o JOURNAL of the AVMA.

Two veterinarians wanted for small animal hospital in eastern state. State your particulars in first letter including starting salary expected. Address "Box W 7," c/o JOURNAL of the AVMA.

Experienced veterinarian wanted for partnership in swine and cattle practice in better section of Middlewest. Prefer age 35 to 40 and graduate of Texas, Kansas, or Colorado. No real estate. Address "Box V 12," c/o JOURNAL of the AVMA.

Nationally established ethical manufacturer distributor of veterinary pharmaceuticals offers immediate opportunity for man qualified and/or experienced in the production of veterinary pharmaceuticals. Address "Box W 5," c/o JOURNAL of the AVMA.

Veterinarian wanted with New York State license for small animal hospital, vicinity New York City. Substantial salary for right man. Address "Box W 8," c/o JOURNAL of the AVMA.

Experienced kennel man wanted for small animal hospital. Address "Box W 15," c/o JOURNAL of the AVMA.

Wanted—Positions

Veterinarian experienced with large and small animals is available for a good position by October. Address "Box W 9," c/o JOURNAL of the AVMA.

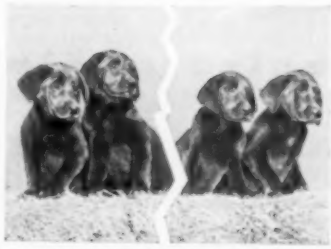
(*Continued on p. 41*)

Continuous maintenance testing proves Friskies fed alone is all a dog needs!

Controlled maintenance tests are among the many being conducted at the Friskies Research Kennels on the Carnation Farms. Mature dogs are fed Friskies exclusively. Only water is added. Many of the tests extend through the dog's full life span. During the tests each dog performs the functions for which the breed is used. For example, a hunting dog spends much time in the field.

FRISKIES-FED DOGS TURN IN TOP PERFORMANCES

At leading dog shows, in the field and just around the house, Friskies-fed dogs are proving the value of a diet that provides every food known to be needed.



SPLIT-LITTER TESTS PROVE FRISKIES A COMPLETE DIET

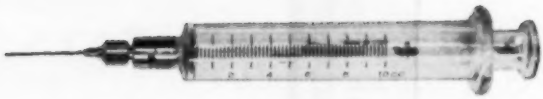
In split-litter tests half the litter is fed on Friskies alone; half, Friskies supplemented with additional foods. In no instance has any benefit been noted from the addition of meat, eggs, milk or vitamin supplements.

Friskies is available in two forms; *Meal*, and bite-size *Cubes*. Both provide complete, balanced nourishment – plus "meaty" flavor dogs love.

SOLD IN 50, 25, 5 AND 2-LB. SIZES

Friskies

ALBERS MILLING CO., DIV. OF
CARNATION COMPANY
LOS ANGELES 36, CALIFORNIA



immunize against distemper and hepatitis with one single shot...

Sin-jex is the first vaccine to utilize a vacuum-dried modified live virus distemper fraction, with killed virus hepatitis fraction as diluent! This modified live virus distemper fraction elicits a more marked response; hence, Sin-jex assures more positive immunity.

R_L's "reconstitiquick" feature means Sin-jex reconstitutes instantly in a fine, homogenous suspension which passes readily through a 22 gauge needle. Sin-jex requires a simple subcutaneous injection which may be given before or after weaning—simultaneously with or without Anti-Canine Distemper Serum and Anti-Infectious Hepatitis Serum (Bival Sera R_L).

Available from independent ethical distributors.

Sold only to graduate veterinarians

Research Laboratories, Inc.
St. Joseph, Missouri

distemper hepatitis
sin-jex
vaccine

(CLASSIFIED ADS—*continued from p. 38*)

Veterinarian with over 2 years of experience desires position leading to partnership, lease, or purchase of mixed practice, preferably in central or south central state. Veteran, married, 2 children; not allergic to work. Address "Box W 3," c/o JOURNAL of the AVMA.

Recent Texas graduate, M.S., D.V.M., wants research position anywhere, preferably on wildlife diseases. Address "Box W 11," c/o JOURNAL of the AVMA.

Experienced veterinarian, 34, married, seeks position leading to partnership or purchase of small animal or mixed practice. Licensed in Ohio, Delaware, Washington, D.C. Address "Box W 12," c/o JOURNAL of the AVMA.

Vienna graduate, 1954 (graduates of this year are recommended by AVMA for recognition by State Board and other agencies) desires assistantship with American veterinarian. Speaks English, age 26, 2 years of experience. Will pay own passage. Address Dr. Egon Schwarz, Bruck an der Leitha, Kirchengasse 11, Austria.

(*Continued on p. 42*)

(CLASSIFIED ADS—*continued from p. 41*)

Wanted—Practices

Small animal practice wanted in New York State; ample capital. Must be a going, established practice completely equipped. Address "Box E 7," c/o JOURNAL of the AVMA.

Want to lease profitable small animal practice, preferably with option to buy, in northern Middlewest; 27, married, experienced, AVMA-approved school. Will consider any good location. Address "Box W 4," c/o JOURNAL of the AVMA.

For Sale or Lease—Practices

Good mixed practice for sale; excellent location, county seat, central state; $6,000 for all drugs and equipment. No real estate. Address "Box W 1," c/o JOURNAL of the AMVA.

Established, operating practice, 90% small animals, for sale; equipped hospital, 8-room residence, and land on main highway in resort area of South. Good for both income and growth; other interests and health. Address "Box W 2," c/o JOURNAL of the AVMA.

Mixed practice, 5 years old, for sale, east central Illinois; modern masonry home and hospital combination, tile office, surgery, etc. Building, 3½ years old, on 1½ acres of land, main highway. No close competition; 65% large, 35% small animals with growth in both. A one-man practice grossing over $20,000. Address "Box W 13," c/o JOURNAL of the AVMA.

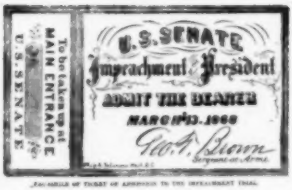
(*Continued on p. 46*)

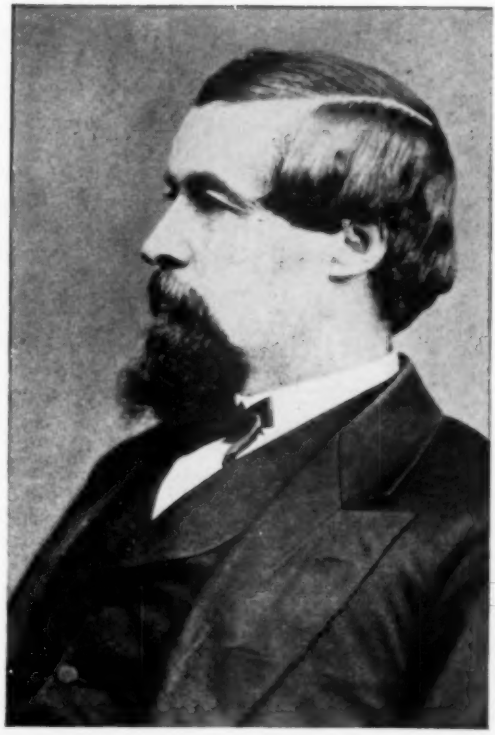
Dr. Robertson Joins Schering Corporation

Dr. William G. Robertson has been appointed veterinary endocrinologist with the Schering Corporation, according to Mr. Robert E. Waterman, vice-president.

Prior to joining Schering, Dr. Robertson completed post-doctoral fellowship work for the Endocrine Society at the Rutgers University Dairy Research Station, Sussex, N. J. In 1951, he received his bachelor of science degree in biology, sciences, and chemistry from Rutgers University and, in 1951, the Ph.D. degree in endocrinology from Rutgers' graduate school.

"I looked down into my open grave"





"MR. SENATOR ROSS, HOW SAY YOU?"

In the deathly silence of the Senate chamber, the freshman Senator from Kansas looked down, as he put it, into his own grave.

On deliberately trumped-up charges, the bitterly fanatic leaders of Ross's party were trying to vote President Johnson out of office—because Johnson stood between them and their revenge on the defeated South.

Ross, they knew, also disliked Johnson and wanted to punish the South. But, taking no chances, they had warned him to vote "guilty" or face political suicide.

And now, on that historic May morning in 1868, the verdict had come to hang completely on his vote. First falteringly, then loudly, he gave it: "*Not guilty!*"

That was sheer moral principle speaking. Edmund Ross refused to join a move he thought would wreck the historic powers of the Presidency. For this, he lost his future, lost his good name, and saved for himself only what he had saved for everyone: our democracy.

Into the whole fabric of American democracy is woven the steel-tough moral fiber of men like Edmund Ross. Braver even than battle courage, it has helped America become strong in many ways. So strong, that, today, one of the world's greatest guarantees of security is U. S. Savings Bonds.

For it is not American principal, but American principles, that back these Bonds. So, for yourself and your country, invest in United States Savings Bonds regularly. And hold on to them.

It's actually easy to save—when you buy Series E Savings Bonds through the Payroll Savings Plan. Once you've signed up at your pay office, your saving is done *for you*. The Bonds you receive pay good interest—3% a year compounded half-yearly when held to maturity. And the longer you hold them, the better your return. Even after maturity, they go on earning 10 years more. So hold on to your Bonds! Join Payroll Savings today—or buy Bonds where you bank.

Safe as America—
U.S. Savings Bonds

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COMPOUND MASTITIS OINTMENT *Lederle*

For professional use and dispensing in Mastitis Control, use an exclusive formula available to veterinarians only

One of eleven dosage forms of POLYOTIC® Tetracycline developed by Lederle for the veterinarian . . . a part of the Red Line of Professional Products for use under your supervision only.

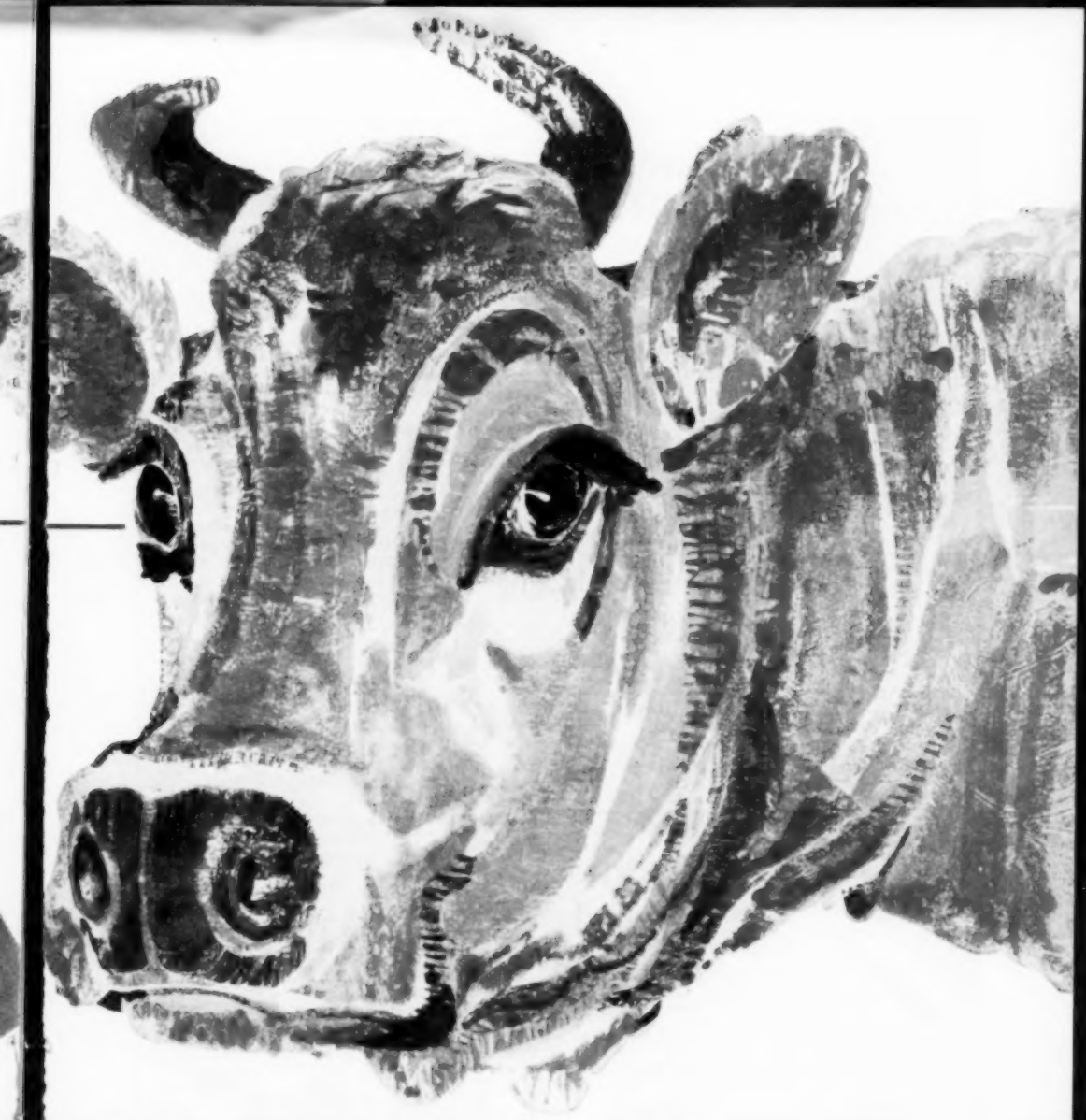
In ¼ ounce applicator tubes. Each tube contains:

Tetracycline HCL	200 mg.
Neomycin	100 mg.
Dihydrostreptomycin	100 mg.
Procaine Penicillin G	200,000 units

. . . in a stable, nonirritating ointment base. May be used in lactating and non-lactating cows.

Lederle

LEDERLE LABORATORIES DIVISION | AMERICAN CYANAMID COMPANY | PEARL RIVER, N. Y.



LEDERLE PROFESSIONAL LINE

POLYOTIC INTRAMUSCULAR:
100 mg.-500mg.-1 Gm.-5Gm.
POLYOTIC INTRAVENOUS: 100 mg.-2.5 Gm.
POLYOTIC OBLETS®: 4's-6 x 4's
POLYOTIC CAPSULES: 50 mg., 25's-100's;
100 mg., 100's; 250 mg., 16's-100's
POLYOTIC TABLETS: 50 mg., 25's-100's;
100 mg., 25's-100's; 250 mg., 16's-100's
POLYOTIC MASTITIS OINTMENT ¼ oz.
POLYOTIC COMPOUND MASTITIS OINTMENT: ¼ oz.
POLYOTIC OPHTHALMIC OINTMENT 1%: 6 x ⅛ oz.
POLYOTIC POWDER 2%: 35 Gm.
POLYOTIC TOPICAL OINTMENT 3%: 1 oz.
POLYOTIC SOLUBLE (Tinted) POWDER:
¼ lb.-½ lb.-1lb.-5 lb.
AVIANIZED® RABIES VACCINE: (Canine):
1 dose-5 x 1 dose-10 doses-100 doses

AVIANIZED RABIES VACCINE: (Cattle): 10 doses
AVIANIZED CANINE DISTEMPER VACCINE:
1 dose-10 x 1 dose
ANTI-CANINE DISTEMPER SERUM AND ANTI-INFECTIOUS
CANINE HEPATITIS SERUM: 100 cc.
INFECTIOUS CANINE HEPATITIS VACCINE: 2 cc.-10 cc.
BRUCELLA ABORTUS VACCINE: 1 dose-5 x 1 dose-
5 doses (25 cc.)
FELINE DISTEMPER VACCINE: 1 immunization (2 vials
Vaccine, 2 vials Sterile Diluent, 2 cc.)
ANTI-FELINE DISTEMPER SERUM: 10 cc.
CARICIDE® Diethylcarbamazine TABLETS:
400 mg., 25's
DIETHYLSTILBESTROL SOLUTION: 10 cc.-50 cc.
LEPTOSPIRA CANICOLA-ICTEROHEMORRHAGIAE BACTERIN
Whole Cultured Inactivated:
1 dose (1 vial Bacterin, 1 vial Diluent, 2 cc.)
Other products to be added.

(CLASSIFIED ADS—*continued from p. 42*)

Busy mixed practice for sale in southern Michigan; new 3-bedroom home, separate modern hospital, both in excellent condition. Priced at real estate value. Address "Box W 6," c/o JOURNAL of the AVMA.

Successful 3-year-old dairy practice for sale in southern Wisconsin. Gross, 1955, $20,000; $6,000 minimum will handle. Price includes house and separate office. Address "Box W 10," c/o JOURNAL of the AVMA.

For sale, to settle estate, veterinary hospital and practice; established over 25 years. Completely equipped including x-ray; outside kennel runs to accommodate 75 dogs. Also 3½-room apartment. Located in one of the most lucrative territories in the United States, southern Connecticut near New York line. A gold mine for a young veterinarian. Address "Box W 16," c/o JOURNAL of the AVMA.

Miscellaneous

Nicholson plasti-plate heating panel provides radiant heat to any kennel for the treatment of shock, postsurgical warmth, weanling puppies, et cetera. Eliminates messy blankets, heating pads, and heat fans. Impervious to chewing, urine, moisture. Panel 23½ in. by 23½ in. with 6 ft. cord only $15.00. Distributed by Professional Products Co., Siesta Key, Sarasota, Fla.

Crisp veterinary medical writing, prompt editorial attention given to manuscripts of all lengths in all specialties, including parasitology, mycology. Consult Scriptors, Box 821, Kalamazoo, Mich.

X-ray Unit for sale—like new and in excellent condition. Profexray Model TC-2 combination radiographic and fluoroscopic examining table with cassette and grid tray, lead-lined drawer and fluoroscopic screen complete. Will give factory new unit guarantee. Ideal for veterinary practice and available at fraction of original cost. Address "Box W 14," c/o JOURNAL of the AVMA.

Artificial Udder—Beco Litter Feeder—nurses 8 puppies simultaneously. Stays warm; easily cleaned and sterilized. Complete unit, $15; guaranteed. Breeders Equipment Co., Flourtown, Pa.

Breedersleve—The disposable obstetrical sleeve. Package of 20 with detachable chest band, $5.00; lower wholesale prices. Free sample upon request. Breeders Equipment Co., Flourtown, Pa.

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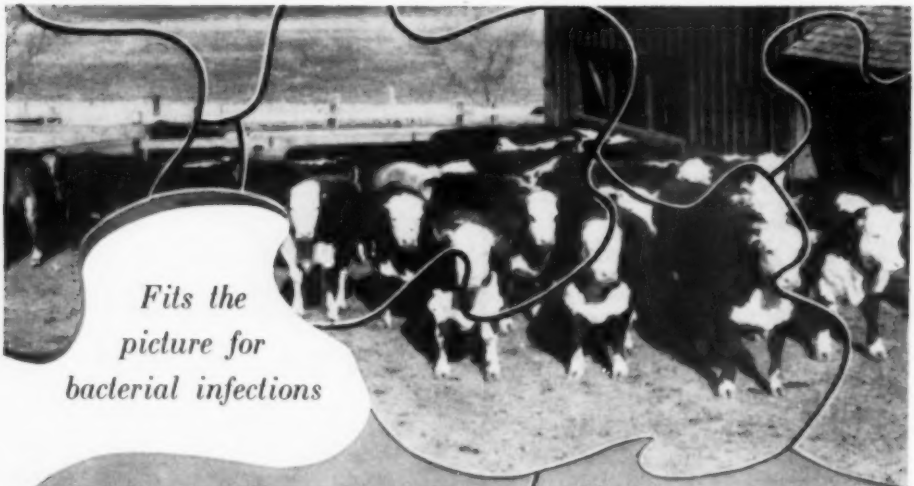
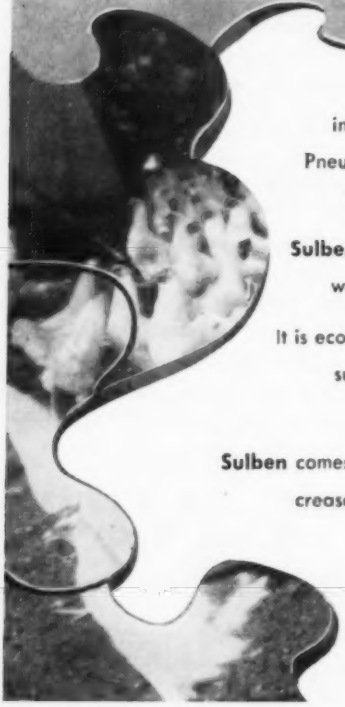


Fits the picture for bacterial infections

SULBEN



Sulben has a wide scope of indications that includes Enteritis, Calf Scours, Hemorrhagic Septicemia, Pneumonia, Foot Rot, Metritis, Mastitis, Coccidiosis, Fowl Cholera, Calf Diphtheria, and many other conditions.

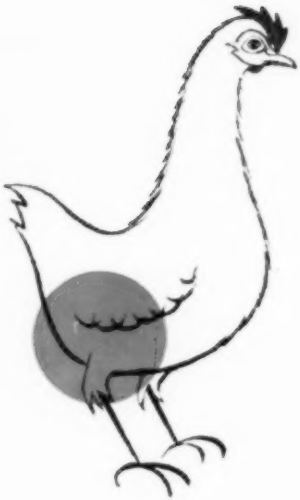
Sulben (sulfabenzamide) is a relatively non-toxic agent, well absorbed, and excreted in a reasonable length of time.

It is economical and dependable. A single dose is usually sufficient, however doses can be repeated every 24 hours or more often if necessary.

Sulben comes in solution for oral use, in 60 gr. creased Tabsules, and in 240 gr. creased Taboles.®

Sulben makes a profitable dispensing item.

HAVER-GLOVER *Laboratories* INCORPORATED



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CHICK EMBRYO ORIGIN

Modified Live Virus Rabies Vaccine prepared from the Flury strain of rabies virus, modified in virulence by passage in chick embryos.

This modified strain has demonstrated high immunizing ability in extensive field trials.

For use only in the immunization of healthy dogs against rabies.

OVINE ORIGIN

A sterile, neutral, phenolized 20% suspension prepared from the brain and spinal cord of animals showing typical symptoms of rabies following subdural injection of the fixed virus of rabies.

For the immunization of dogs and other animals against rabies.

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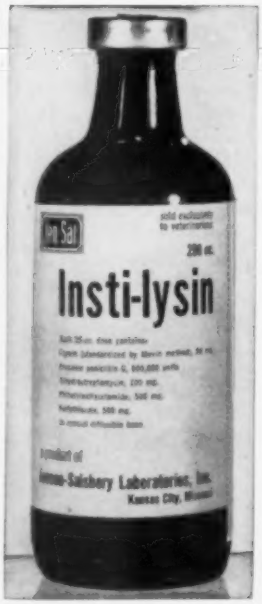

each 25 cc. dose
contains
50 mg. Papain
(pure crystalline)
500,000 units
Procaine penicillin G,
100 mg.
Dihydrostreptomycin,
500 mg.
Phthalylsulfacetamide,
500 mg.
Sulfathiazole,
in special
diffusible
base

pure crystalline Papain
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the superior therapy in acute mastitis

The superior effectiveness of Insti-lysin as therapy in acute mastitis and other conditions requiring effective debridement has been well established by continuous field usage.

Pure crystalline papain gives Insti-lysin twice the proteolytic activity of crude papain, without irritation to normal tissue, and without systemic or generalized reactions.

Antibiotics and sulfonamides assure broad bacterial coverage and maximum antibacterial activity. As an udder infusion, Insti-lysin rises rapidly to the site of infected tissue in its special, diffusible base.

Insti-lysin is available in 3 different packages to meet the specific needs of individual practices; 250 cc. vial for professional use; display carton of 12—28 cc. mastitis syringes in individual dispensing cartons; package of 12—5 cc. syringes in dispensing cartons for small animal clients.

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